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### STAPHYLOCOCCAL PNEUMONIA IN INFANCY AND CHILDHOOD.<sup>1</sup>

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THE object of this paper is to discuss a group of 32 infants and children suffering from staphylococcal pneumonia, who were admitted to the Royal Alexandra Hospital for Children in the two-year period from December, 1954, to November, 1956. This group comprised 28 proven cases, in which the diagnosis was established by growing on culture *Staphylococcus aureus* coagulase-positive (hereafter referred to as *Staph. aureus*) from the pleura or lung, and four probable cases with clinical and radiological features typical of staphylococcal pneumonia, and a profuse growth of *Staph. aureus* on culture of a laryngeal swab. Excluded from the group were 13 cases of uncomplicated pneumonia in which laryngeal cultures gave positive results. Also excluded were six cases in which staphylococcal pneumonia was a terminal complication of kernicterus, cystic fibrosis of the pancreas or gross congenital cardiac or other defects.

#### Age, Sex and Seasonal Incidence.

Of the 32 children, the ages of 12 were less than three months, of 19 less than six months and of 28 less than two years. Thus

<sup>1</sup> Read at a meeting of the Australian Paediatric Association, Canberra, March 30 to April 1, 1957.

only four children in the series were aged two years or more (Table I). The oldest child was aged seven years, and the youngest 16 days. The average age on admission to hospital of the group of infants aged less than three months was 40 days. Sixteen of the children were males and 16 females, an even distribution between the sexes occurring at each age period.

The incidence was higher during the winter, as 25 of the children were admitted to hospital between April and September, and only seven in the six-month period from October to March (Figure 1). The peak occurred with seven admissions during the month of August, and it is interesting to note that no cases were admitted during January or February of the years under consideration. Twenty children were admitted during the second 12 months, compared with 12 children during the first 12 months.

#### Predisposing Factors.

Many of the infants aged less than three months had a history of abnormal delivery, low birth weight, or poor development *post partum*. Mechanical difficulties occurred during the birth of four infants, and the birth weight of two was less than five pounds. Five infants suffered from feeding difficulties or failed to gain weight normally, and only two of the 12 were considered to have had a normal birth and normal development until the onset of signs of pneumonia. In the group aged three months or more, these non-infective contributory factors were not noted, and specific infective diseases—measles and varicella respectively—preceded the onset of pneumonia in only two children. However, there was frequently a history of a local epidemic of influenza, or

of a member of the immediate family having recently suffered from this disease.

#### History of Onset.

The respiratory disease, in 26 of the 32 children, began with the simultaneous appearance of coryza and cough; the gradual progression from coryza to cough described in other series was not noted. In 22 of these 26 children, such signs of pneumonia as fever, cyanosis, tachypnea and grunting, laboured respirations developed after an average interval of eight and a half days. Coryza and cough progressed to abdominal distension, with or without anorexia, vomiting or constipation in four children, each of whom was provisionally diagnosed on admission to hospital as suffering from an intraabdominal condition. Staphylococcal skin infection preceding the onset of pneumonia was recorded in the histories of three children. One infant suffering from extensive osteomyelitis of the mandible died suddenly, and staphylococcal pneumonia with lung abscess and empyema, unsuspected during life, was found at post-mortem examination.

TABLE I.  
Age Incidence and Mortality in 32 Cases of Staphylococcal Pneumonia in Childhood.

Age.	Number of Cases.	Number of Deaths.
Less than three months	12	4
Three to six months	7	3
Six months to two years	9	1
Two years and over	4	1
Total	32	9

#### Physical and Radiological Signs.

The physical and radiological signs on admission to hospital were, in many cases, non-specific and non-localizing (Table II). Tachypnoea in 26 cases and fever in 21 were the most commonly recorded signs, whereas impairment of the percussion note and diminished respiratory movement of the chest were noted in approximately half of all cases. Abnormalities in the intensity or quality of the breath sounds were noted in 20 cases. Adventitious were heard in 15 cases, but the distribution had localizing value in only seven of these. Cyanosis was observed in nine cases, pallor in six and "collapse" in five. Abdominal distension occurred in eight cases, comprising the four already mentioned, in which it dominated the clinical picture, and four in which it was present in addition to signs referable to the respiratory system.

In 22 of the 32 cases, radiological examination of the patient on admission to hospital revealed opacities suggestive of consolidation, consolidation and fluid, or fluid alone (Table III). Air cysts, pneumothorax and pyopneumothorax were noted on admission to hospital in only seven cases.

#### Bacteriological Diagnosis.

The in-vitro sensitivity to antibiotics of the first culture of *Staph. aureus* from the pleura or lung was estimated in 25 cases by means of the disk method, using the Evans "Sentest" disks. The organism was sensitive to penicillin in two, to the tetracyclines in 22, and to streptomycin and chloramphenicol in each of the 25 cases. Thus, in only two of the 25 cases was sensitivity to penicillin demonstrated. The organism was sensitive to erythromycin in all cases (about one-third of the total number) in which this sensitivity was determined.

#### Mortality.

The mortality was much higher in infants aged less than six months (Table I). Of the nine children who died, seven were aged less than six months, one was aged six months and one was aged two years. This represents an overall mortality of nine out of 32, and a mortality in the first six months of life of seven out of 19. In this group of fatal cases the average time interval between the onset of frank signs of pneumonia and death was five days, and between admission to hospital and death, two days.

#### Management.

Careful attention was given to general and supportive therapy. Every effort was made to achieve adequate periods of undisturbed rest by reducing unnecessary handling to a minimum, by tube feeding if sucking exhausted the infant, by the grouping of disturbing procedures, and by adequately sedating the child with phenobarbital or chloral hydrate. Oxygen was administered when necessary, and attempts were made to place the child in the position of greatest comfort.

Anæmia developed in about one-third of the children, often with surprising rapidity, and was corrected by small transfusions of packed cells, administered slowly, and repeated if necessary. These transfusions often appeared to have a favourable effect on the course of a child's illness, greater than might have been expected from the correction of anæmia alone.

#### SEASONAL INCIDENCE

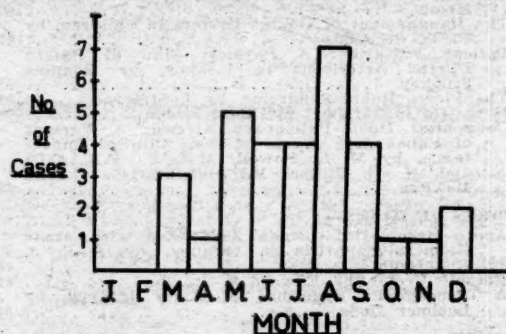


FIGURE I.

Seasonal incidence in 32 cases of staphylococcal pneumonia in patients admitted to hospital in the two-year period from December, 1954, to November, 1956.

Changes were observed in the trends of antibiotic therapy during the two-year period. In the early months, penicillin was often used, reliance was frequently placed on a single antibiotic, and repeated changes were often made in antibiotic therapy as a child's condition deteriorated. With increasing awareness of the high incidence and fulminating nature of the disease, it became the tendency to administer a combination of two broad-spectrum antibiotics to infants with clinical and radiological signs of pneumonia. Chloramphenicol or one of the tetracycline group was usually combined with streptomycin.

Complications resulting from the administration of broad-spectrum antibiotics occasionally proved a problem. Complements of the vitamin B group were given as a routine procedure, to offset any diminution in production due to altered bowel flora. Looseness of the stools, which not infrequently occurred, was not in itself regarded as an indication to suspend an antibiotic. However, if diarrhoea was severe or persistent, cultures of the stools were attempted, to detect a superimposed staphylococcus enteritis, nystatin (when it became available) was administered to deal with any monilial infection, and it was occasionally necessary to change the antibiotic or administer it by the parenteral route. In an attempt to minimize the hazard of bone-marrow depression by chloramphenicol, white-cell counts were made weekly. No evidence of bone-marrow depression was noted in any case in this small series.

A constant watch was kept for the onset of intrathoracic complications, and repeated clinical and radiological examination was regarded as essential. Despite the hazard of excessive radiation and the child's need for rest, physical examination and radiography were not deferred for these reasons alone. Even when under tension, pneumothorax in small infants was often detected only by radiographic examination. For this reason a chest X-ray examination was made whenever there was sudden, unexplained deterioration in the condition of a child with this disease.

#### Discussion.

Study of the present series thus illustrates the fact that staphylococcal pneumonia is predominantly a disease of infancy, with its greatest incidence and mortality in the first six months of

life; that there is frequently a warning period of mild symptoms referable to the upper part of the respiratory tract, but that, with the development of frank signs of pneumonia, there may be rapid progression to complications and death; and that, at the time of the infant's admission to hospital, there is often neither clinical nor radiological evidence that the disease is due to *Staph. aureus*.

However, several problems require further discussion. First, it is difficult to determine the overall incidence of staphylococcal pneumonia, as only those cases in which *Staph. aureus* is obtained from the blood, pleura or lung may be confidently included in a series. Certainly, a pure growth of an organism from culture of a laryngeal swab suggests that this is the causative agent; but a pure growth of *Staph. aureus* may be obtained from the larynx of normal persons.

TABLE II

Physical Signs Noted on Admission to Hospital in 32 Cases of Staphylococcal Pneumonia.

Sign.	Number of Cases.
Increased respiratory rate .. ..	26
Fever .. ..	21
Impaired percussion note .. ..	16
Diminished inspiratory movement ..	12
Breath sounds:	
Diminished .. ..	15
"Bronchial" .. ..	5
Adventitious:	
Generalized .. ..	6
Localizing .. ..	7
Non-localizing .. ..	2
Cyanosis .. ..	9
Pallor .. ..	6
"Collapse" .. ..	5
Abdominal distension .. ..	8

Pleural aspiration is performed if there is clinical and radiological evidence of effusion; but these signs are absent in most cases on admission to hospital. Recently, successful use has been made of the technique of lung puncture, described by Disney *et alii* (1956), which differs from routine chest aspiration in that a thin 18 or 20 gauge needle is employed. If no exudate is obtained from the pleural cavity, the needle is advanced a further centimetre or two into the lung, suction being maintained during both insertion and withdrawal. If visible exudate is seen in the syringe, it is inoculated on to a blood-agar plate. If no exudate is obtained, the needle is inserted into a test-tube containing sterile glucose broth, which is sucked up into the syringe, discharged back into the tube, and then cultured. The technique was recently performed in two cases not included in the series, and pure growths of pneumococcus and *Staph. aureus* respectively were obtained from culture of the lung juice. This modification is not recommended as a routine procedure, but is valuable in cases with little or no pleural exudate, in which aspiration is being carried out for purely diagnostic purposes. The corollary is that after pleural tap, even if this is unsuccessful, glucose broth should be drawn up into the syringe and then cultured.

Blood culture may yield the organism if there are signs suggesting blood-stream invasion, but is usually abandoned if superficial veins are scanty and attempts to obtain blood are likely to distress the child unduly.

The second problem is the evaluation of predisposing factors and their differentiation from early manifestations of the disease. It is well known that staphylococcal pneumonia frequently complicates cystic fibrosis of the pancreas, and may be a terminal complication in grossly debilitated children. But what of the child who fails to thrive from birth and dies at the age of two months from staphylococcal pneumonia? Has he acquired a staphylococcal infection in the nursery, which retards his progress and finally manifests itself as a fulminating pneumonia? Or is his illness a cross-infection acquired at the age of seven weeks by a debilitated infant in hospital? It is often impossible to determine the answer to such a question. Furthermore, the initial episode of coryza and cough is generally regarded as representing a viral infection of the upper part of the respiratory tract, paving the way for the entry of the staphylococcus. The

seasonal incidence of the disease, and its known tendency to occur in a community which is sustaining an influenza epidemic, support this hypothesis.

Abdominal distension, which has often been interpreted as a sign of some predisposing condition, is now recognized as a frequent manifestation of the disease. It is caused by gaseous distension of the bowel, thought to be due to toxic paralytic ileus. Thus, the rapid onset of abdominal distension, particularly if it is preceded by coryza and cough, or if it is accompanied by fever and respiratory distress, strongly suggests the possibility of staphylococcal pneumonia.

The third problem is the determination of a rational approach to antibiotic therapy. Apart from the obvious advantage of early intensive therapy with broad-spectrum antibiotics, no conclusions can be drawn from the present series as to the best

TABLE III

Radiological Signs Noted on Admission to Hospital in 32 Cases of Staphylococcal Pneumonia.

Condition Suggested by Chest X-Ray Appearances.	Number of Cases.
Consolidation .. ..	12
Consolidation and effusion .. ..	5
Effusion .. ..	5
Tension of pneumothorax .. ..	4
Air cysts not under tension .. ..	1
Air cysts under tension .. ..	1
Loculated hydropneumothorax .. ..	1
No abnormality .. ..	1
X-ray examination of chest not performed ..	2
Total .. ..	32

régime. However, certain general principles may be laid down. First, penicillin has no place in the initial management of staphylococcal pneumonia, and should be given only if and when in-vitro sensitivity to the drug has been demonstrated. Second, it is probably wisest to use a combination of at least two antibiotics to combat this severe infection, and to reduce the likelihood of insensitivity to therapy. While the combination of chloramphenicol or a tetracycline with streptomycin provides a satisfactory régime in cases of moderate severity, erythromycin appears to be particularly indicated in the management of infants in the first three months of life, and of older children with severe toxæmia. In such cases the combination of erythromycin and chloramphenicol, with or without streptomycin, is probably the most effective method of treatment.

The value of intensive antibiotic therapy is greatest in the first few days, in controlling toxæmia and preventing the onset of complications. The parenteral route is therefore used for at least the first 12 hours in order to obtain high blood levels as rapidly as possible. If circulatory collapse is present, a broad-spectrum antibiotic is given intravenously. Antibiotics are administered in maximal dosage at the commencement of treatment, this being reduced when there is adequate evidence of clinical response. Antibiotic therapy appears to be less effective in eradicating the disease than in the early control of toxæmia, and it will frequently be necessary to continue the administration of antibiotics for many weeks.

The final problem to be discussed is the early management of pneumonia in infancy before a bacteriological diagnosis has been established. Disney *et alii* (1956) make the following statement:

It therefore seems that the first definite sign of localised pneumonia in infants aged less than two years demands immediate treatment as a potential staphylococcal infection.

The present series bears out this suggestion. Staphylococcal pneumonia in infancy may be a fulminating disease with rapid progression to complications and death. The results of culture and sensitivity studies are not available for at least 24 hours after the patient's admission to hospital. It therefore seems reasonable to agree that any infant aged less than two years with clinical and radiological signs of pneumonia should be treated as suffering from staphylococcal pneumonia, unless or until there is bacteriological evidence that *Staph. aureus* is not the causative agent. A more conservative approach may be adopted to the therapy of a child aged more than two years; but if an older child appears seriously ill or fails to respond to a conservative régime, antibiotic therapy, as for staphylococcal pneumonia, should be immediately commenced.

### Conclusion.

In conclusion, it is suggested that the mortality and morbidity of staphylococcal pneumonia will be reduced only by regarding any case of pneumonia in infancy as potentially staphylococcal in origin, by making every effort to determine the nature and sensitivity of the causative organisms, by immediate institution of intensive antibiotic therapy, and by careful clinical and radiological observation of the patient in hospital to ensure the early detection and prompt treatment of complications.

### Acknowledgements.

I wish to thank the Honorary Medical Officers of the Royal Alexandra Hospital for Children for their cooperation in enabling me to study the children under their care.

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## CYSTIC DISEASE OF THE LUNGS IN CHILDHOOD.<sup>1</sup>

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THERE are many conditions affecting the lungs in childhood which may give rise to a cystic appearance radiologically. To mention a few of these, cysts may develop in association with staphylococcal pneumonia, cytomegalic inclusion disease, tuberculous sclerosis, hydatid disease, Hands-Schüller-Christian syndrome, etc. However, this paper deals mainly with the group which have been labelled as congenital cysts of the lung, and, as in the acquired variety, such congenital cysts may be single or multiple.

There has been considerable discussion in the literature as to the exact definition of a congenital cyst, and there appears to be much confusion with no really fixed ideas concerning this classification. It has been stated frequently that it is essential for a congenital lung cyst to be lined by bronchial epithelium, and that, if the lining is one of alveolar cells, then the cyst is no longer congenital. This statement appears to imply that all cysts are bronchial in origin. However, whilst bronchial cysts are undoubtedly the commonest, cysts of alveolar origin do also occur, and furthermore, many so-called congenital cysts of bronchial origin are really acquired—i.e., secondary to infection.

A congenital lung cyst may be defined as one arising from some maldevelopment of the bronchi or alveoli and a satisfactory classification is (i) bronchial, (ii) alveolar. Each of these types may or may not communicate with the normal bronchial tree.

### Bronchial Cysts.

In the development of the lung buds from the primitive foregut of the embryo there may be arrest or distortion of development at any point. If this occurs in a proximal portion of the lung bud, there will result either a sequestered pulmonary lobe or a bronchogenic mediastinal cyst.

Thus the first type of congenital bronchial cyst is that which has been described as "congenital cystic disease of the lung associated with anomalous arterial distribution",

or perhaps more simply as a "sequestration of the lobe of the lung". One can classify this as truly congenital, for it owes its origin to a developmental abnormality.

This condition was described by Fischer in 1928, but was first detailed adequately by D. M. Pryce in 1946. The fundamental abnormality is that the arterial supply is derived from the aorta, either thoracic or abdominal, there being no pulmonary artery to the affected segment, and in addition there is a complete dislocation of the bronchus from the main bronchial tree in that part supplied by the abnormal artery. The artery which is derived from the aorta is still histologically a pulmonary artery and is subject to early atheromatous changes.

It is considered by Pryce that the arterial abnormality is primary; he postulates that during the lung development the bulbous tip of one branch of the primitive bronchus may be captured by portion of the dorsal capillary plexus, and subsequent development causes its distortion and complete dislocation. The amount of separation from the ordinary lung tissue may be partial or complete, thus giving rise to the terms intralobar and extralobar sequestration; in the latter case the sequestered lobe has been described even within the abdominal cavity. These cysts cause no trouble unless they become secondarily infected by hematogenous or contiguous spread, when they produce symptoms suggestive of bronchiectasis and are usually cured relatively simply by lobectomy.

If the arrested or distorted development of the lung bud occurs more peripherally, cysts will be found within the otherwise normal lung tissue. Such cysts have an epithelial lining consisting of columnar or cuboidal cells, which may or may not be ciliated. The wall contains the normal bronchial elements, but these elements—viz., the cartilage, smooth muscle, elastic tissue and mucous glands—are not in the usual orderly arrangement of the normal bronchus.

These cysts become manifest clinically if one or two of the following events occur: (a) infection, when they cause symptoms similar to those of bronchiectasis; (b) rapid increase in size, when they cause respiratory distress, severe dyspnoea and cyanosis. On examination of the patient in the latter case there are diminished breath sounds and hyperresonance on the affected side, with mediastinal shift to the opposite side. Such cysts are confined usually to one lobe or portion of one lobe, and are shown radiologically as clear air-containing spaces divided by septa into large segments with the adjacent lobe or lobes compressed.

Lobectomy or pneumonectomy is usually required for adequate treatment, but sometimes the primitive bud from which the cyst has developed is so separated from the rest of the lung that the cyst can be removed by ligation of its pedicle.

For such a sequence of events to occur there must be a communication with the bronchial tree, and the sudden distension is due to a ball-valve mechanism similar to a valvular pneumothorax. This occurs especially in infancy because, owing to the small calibre of the bronchial tube and the weakness of the wall at this age, such a mechanism is produced more easily. However, not all bronchial cysts communicate with the bronchial tree, and in such cases the main branches of the bronchi may end abruptly and blindly in the development of cysts.

Pathologically, the wall structure is similar to that of the communicating type of cyst; but here there has been apparently marked segmentation of the primitive lung bud followed by subsequent dilatation.

Not all radiological cysts of bronchial origin are congenital. To illustrate this point I should like to present a very interesting case which we have watched at the Royal Alexandra Hospital for Children, Sydney, for some time.

The patient is a boy, aged eight years. From the neonatal period until the age of two and a half years he suffered recurrent attacks of fever of unknown origin. A broncho-

<sup>1</sup> Read at a meeting of the Australian Paediatric Association, Canberra, March 30 to April 1, 1957.

gram taken at this age, on March 1, 1951, was of normal appearance, and the whole lung field appeared normal (Figure I). At the age of two and a half years he commenced to have attacks of cough, productive of sputum, and fever, which lasted three or four days; these febrile attacks occurred regularly at monthly intervals. Gradually the attacks became more severe, and the sputum became offensive and yellow and was present in large amounts. An X-ray examination of the chest made on February 23, 1955, showed cyst-like cavities in both lung fields, some containing fluid (Figure II). A bronchogram taken on March 17, 1955, showed that these cystic lesions, which were bilateral, communicated with the bronchial tree (Figure III). The cysts enlarged, and on July 24, 1956, they were gross, with multiple fluid levels (Figure IV). At this stage it appeared that the child was carrying on with a partial compression of the upper lobe of the right lung and of portion of the upper lobe of the left lung. As the child's condition was deteriorating it was decided on August 1 to excise the diseased segments, commencing with the right side. On September 17 this was done by Mr. E. Stuckey, and at operation it was found that the cystic condition was confined to the middle lobe of the right lung and to the anterior basal segment of the lower lobe of the right lung. There was an abnormal arterial distribution of the pulmonary artery, the affected segment of the lower lobe of the right lung being supplied by the artery to the middle lobe of the right lung.

Pathological examination carried out by Dr. R. D. K. Reye was reported on as follows:

The specimen consists of the entire right middle lobe and a segment (anterior basal) of the right lower lobe. The right middle lobe is larger than normal and to palpation it is apparent that it is composed largely of cysts.

After fixation the lung tissue was explored by opening into the main bronchi at the hilum. A short distance from the hilum the bronchi opened into a series of large cysts all of which communicated with the bronchial tree, though in all areas of bronchial bifurcation the openings into the cysts were narrow and valve-like. The cysts appeared to have developed as gross sacular dilatations of all the small branches of the main bronchi.

In the upper parts of the lower lobe the main bronchus divides into one branch which passes into a collection of large cysts while two other branches are merely dilated in a cylindrical manner.

Microscopic: Sections of various parts reveal a little compressed lung tissue between the cysts, while the cysts are lined by bronchial mucosa and have walls which contain a little smooth muscle but which are largely fibrous. A striking feature is the entire absence of cartilage in the walls of any of the cysts examined; cartilage was found only in the main bronchi close to the hilum.

Comment: Though a congenital cystic maldevelopment of the bronchi cannot be excluded with certainty, the fact that all of the cysts are in communication with the bronchial tree and can be traced as branches of the main bronchi seems to favour a sacular type of bronchiectasis of unusual severity. It is possible that the unusual degree of dilatation rests upon a congenital defect and that there is a deficiency in cartilaginous support to the wall. However, the simple failure to locate cartilage in sections from the grossly dilated bronchus could be a mere matter of chance and so absolute proof of such a congenital deficiency in this case cannot be said to have been fully established.

The point to be stressed is that this case was diagnosed confidently by the radiologist as being one of congenital cystic disease of the lung, it being unknown at that time that normal bronchographic findings had been obtained and that an X-ray film of the chest had shown normal appearances.

This mistake must have been made on many occasions in the past when no X-ray films of the chest have been taken prior to the onset of infection.

Apart from this case, I have now in my care three other children with a similar diagnosis, from whom I have been able to obtain a normal X-ray picture prior to infection.

These cysts appear to depend upon some deficient development of the walls of the bronchi—e.g., deficient cartilage—and this weakness does not become manifest

until a superadded infection leads to further weakening. My own thoughts are that this is well described by the term "congenital bronchomalacia", as used by Engel in his book "The Child's Lung".

To illustrate the difficulty in deciding whether a cyst is congenital or acquired after infection has occurred it is necessary only to refer to the following two commonly used criteria of congenital origin: (i) An epithelial lining of the cysts is stated to signify such an origin. However, this is not valid, as after destruction of pulmonary tissue one frequently sees epithelial regeneration, as in the lining of the large sacculi of saccular bronchiectasis or in lung abscess. (ii) The presence of cartilage in the walls of a cyst has been taken as proof of a congenital origin; but again, cartilage is frequently found in the walls of sacculi in saccular bronchiectasis.

### Alveolar Cysts.

As in the case of bronchial cysts, alveolar cysts may be congenital or acquired. In the acquired type, the development is due to bronchiolar obstruction produced by infection, which permits the entry of air during inspiration, but prevents the exit during the phase of expiration.

As a consequence, the alveoli distal to the obstruction become distended, with some breakdown and the formation of a pseudocyst. The lining of the cysts is one of matted alveolar cells.

In the congenital type there is a maldevelopment of the primitive alveolar bud, but this occurs at a later stage of development than in cysts of bronchial origin. Such cysts are not usually present at birth, but become distended during post-natal respiration. These cysts, which are lined with epithelium and may contain mucoid fluid, pus and air or desquamated debris, may or may not communicate with the bronchial tree. If such communication is present, and especially if infection is also present, there may be increased accumulation of air with the production of tension, as in cysts of bronchial origin.

### Discussion.

In this paper an attempt has been made to correlate the various types of congenital lung cysts with an abnormality of the development of the primitive lung bud at various stages.

At present the greatest doubt in any particular case must exist in deciding whether those bronchial cysts communicating with the normal bronchial tree are congenital or are acquired as a result of infection. It is to be noted that most of these types of congenital cysts are not apparent clinically until such infection has taken place. However, it is thought that too often in the past such cysts have been labelled congenital without any satisfactory basis for such a diagnosis.

### Acknowledgements.

I wish to thank Professor Lorimer Dods for permission to discuss this case, and Dr. R. D. K. Reye for the pathological report.

### References.

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### Legends to Illustrations.

- FIGURE I.—Norman bronchogram, March 1, 1951.  
 FIGURE II.—Skilogram of the chest, February 23, 1955, showing large bilateral air cysts.  
 FIGURE III.—Bronchogram, March 17, 1955, showing air cyst filled with "Lipiodol".  
 FIGURE IV.—Further development of air cysts, some with fluid level. (July 24, 1956.)

READING DISORDERS IN CHILDHOOD.<sup>1</sup>

By ELIZABETH A. KEMP.

READING being the foundation of our educational system, failure or delay in learning to read can mean limitation of the child's opportunities, and a prolonged experience of inadequacy which can have serious effects upon his mental health.

It has been estimated that approximately 12% to 16% of the school population has difficulty in learning to read. To understand the problem, many have devoted a lifetime of investigation, contributing to an extensive body of literature on the nature of the learning process, the factors underlying readiness for reading, appropriate experience programmes and materials, and the recognition, diagnosis and management of special learning problems. For comprehension of what might be called the pathology of reading, the teacher has turned to the medical profession for explanation and advice; under certain very favourable circumstances it has been possible to bring together, for this purpose, the paediatrician, the neurologist, the ophthalmologist, the hearing specialist, the speech specialist, the psychiatrist, the endocrinologist, the social worker, the psychologist and the teacher. Development of the school medical services within the Commonwealth has been of paramount importance in drawing attention to handicaps and anticipating difficulties, while the vigilance of the various Child Welfare Departments and social services will continue not only to improve the conditions surrounding the child's nurture, but also to draw to the attention of the administration unsatisfactory conditions of housing, finance etc., which may handicap the development of the junior citizen.

Investigation of failure is one of the routine functions of the various guidance services within the Commonwealth. A recent survey in a group of schools within the metropolitan area of Sydney showed, among other degrees of failure, that one child in 85 was so seriously retarded in reading as to be functioning at a level two years or more in arrears of his assessed mental age. It is thought that this estimate of very severe retardation is conservative, and that it does not in this sample reflect an unusual concentration of reading problems in the schools concerned. The usual combinations of underlying difficulties were discerned from individual case studies—namely, health disturbances, physical and intellectual handicaps, sensory defects, disturbed schooling, social, domestic and environmental problems, premature introduction to formal instruction, and so on.

While bearing in mind that learning failure is usually associated with a complex set of conditions, I should like to draw attention to three relatively distinct types of problem, confining discussion to the child who apparently cannot learn as readily as his fellows, excluding for this purpose the child who rejects learning, or is prevented from learning. The first, and numerically strongest type, is exemplified in the person of Robert.

Robert is the only son, with a bright younger sister, of a materially comfortable family, the parents having plans for expensive private education in a predominantly grammar-type school. He has enjoyed good health, early medical attention when necessary, regular school attendance, and the care of parents who are as vigilant of his needs as they know how to be. However, the parents do not fully share the understanding of the school that Robert is a somewhat slowly developing child, and much pressure has been exerted upon him at home to do better. He is growing up with feelings of guilt, failure and inadequacy, and time which might be spent more profitably in the building of practical skills is being devoted to academic experiences he is not ready to assimilate. Despite a somewhat superior speaking vocabulary and a well-disciplined demeanour, which tend to mislead the casual observer into overlooking his intellectual limitations, Robert's powers of judgement, reasoning and discrimination are at a level younger than his chronological age. As evidence of this, when he was ten and a half years old, he was trying to cope with the educational programme designed for the child of his chronological age, with the understanding of a nine year old, and the reading skill of a seven year old.

Since his reading skill was so far in arrears of his apparent ability, the possibility of specific reading disability was raised,

but it was thought that Robert's difficulties could be explained in other terms. It is a common observation that children do not normally begin to respond to experience in early reading until they have attained the mental age of six to six and a half years, other conditions, physical, emotional and environmental, being reasonably satisfactory. It would be inferred that Robert, in common with tens of thousands of other slow-learners, would have entered first grade at a stage when he was too immature to derive full benefit from the experience offered, and steady promotion from grade to grade would have served only to aggravate the educational problem.

When academic progress is slow and laborious, some parents are realistic in providing opportunities for the child to develop functions other than those associated with book-learning. Robert's parents place a premium on academic education, and will continue to find it difficult, if not impossible, to reduce their absorption in the more formal aspects of schooling, or to assist the lad in finding acceptable alternatives. They have much in common with the parents of Bill, who has been promised the tools he craves only when his reading and spelling show very marked improvement, and with the parents of Peter, who is denied valuable opportunities for social experience by playing with his friends after school, because he must spend extra time with his lessons.

It has been estimated that approximately 15% of the child population belong to this slightly slow group. There are, of course, many children slower than Robert, and others like him, but these are usually clearly recognized as mentally much younger and less capable than others of their age. The Roberts in the community are often those most seriously misunderstood, because their learning difficulties are comparatively mild in degree. Many of these children react to the demands made upon them with resentment, indifference, hostility, rebellion, or withdrawal from a way of life which attempts to constrain them into a mould for which they are not fitted.

Ideally, in a school community fully served by a guidance programme, it would be possible to identify these children at kindergarten level, and thereby ensure that they were not exposed to formal learning experiences before they were ready. In many cases, however, understanding of their limitations occurs when the pattern of failure has already been established.

A different type of problem, that of a generalized language disability, is exemplified by Tom, now 11 years old, apologetic, unsure of himself, self-conscious, groping for words, grammatically confused, having difficulty even in recalling the names of familiar objects. This lad does fairly well at the practical level, showing resourcefulness and initiative; but he is at a disadvantage in expressing himself in words, and scores below average in tests of judgement and reasoning couched in verbal terms. As would be expected, his reading skill is much in arrears of his age. A much more serious degree of the problem is exhibited by Brenda, who, we are told, was intellectually normal until the age of five years, when she suffered a childhood infection which left brain damage with residual aphasia. Special testing showed soundly normal development on the non-verbal achievement level, but as people are frequently accepted or rejected on their verbal facility, Brenda finds herself somewhat isolated by her age group, who consider her response to verbal communication and her level of conversation exasperatingly childish. Brenda's reading, which is at the level of the average child six and a half to seven years of age, at the chronological age of fourteen and a half years, is no better than would be anticipated from her level of verbal development.

Now that our measuring devices are becoming more precise, we are finding many children who are below average in language development, despite evidence of ability at average or superior level in other aspects of growth. The incidence of this type of problem is obscure; but there is reason to believe that mild degrees of immaturity in the use and understanding of spoken language could remain unrecognized and uncompensated. The problem awaits further study.

In the third group of children there is demonstrated such a profound degree of difficulty that they appear to suffer from some kind of mental crippling. A concentration of these subjects would be referred to any child guidance or educational clinic service; for example, in the clinic with which I am associated, of a total of 700 children tested individually during 1956, it is suspected that 45 were suffering from some form of reading disability. The incidence of the problem in the community

<sup>1</sup> Read at a meeting of the Australian Paediatric Association, Canberra, March 30 to April 1, 1957.

cannot be estimated with any confidence, but it seems possible that 5 to 10 in every 1000 would not be an over-estimate.

The presenting problem is usually that of poor school progress, not always recognized as stemming from inadequate reading skill. A striking feature is that many children of otherwise soundly normal, or even apparently superior, understanding are affected. Features of individual response to diagnostic tests are as follows: (i) reading skill far below the level of the mental age, and seriously at odds with the schoolwork expected of the child; (ii) extremely slow and laboured word recognition, and a lack of understanding of appropriate means of attack on unfamiliar words; (iii) difficulties in auditory discrimination or recall of sounds and sound combinations, not associated with deafness; (iv) difficulties in discrimination and recall of visually presented shapes and patterns; (v) persistent difficulties in maintaining the conventional directional attack in word recognition.

Overseas literature suggests that at least twice as many boys as girls are affected by this disorder; in our clinic, during 1956, the proportions were even more strongly to the disadvantage of the boys. It is noted, too, that even when other possible factors have been eliminated, the disability sometimes affects more than one member of the family. The occasional association of such cases with a reported history of instrumental birth, birth damage, Rh incompatibility, brain damage following illness or injury, pink disease, epilepsy and the like, has been noted, whether significantly or not.

When the disability is profound, the child seems to have little or no recollection of a word to which his attention must have been drawn hundreds of times. This characteristically fugitive memory for word patterns is the feature which parents and teachers find so difficult to understand, and in far too many cases the criticism and nagging to which the child is subjected must achieve nothing but a most mischievous result in his treatment.

It is realized that many attempts have been made to explain the learning problems of these children as arising from some form of cerebral defect or delayed maturation. Considerable significance has been attached in the past to the observation that in many of these cases there is associated a condition of confused laterality; but many investigators tend to agree with Gates, who states that "left handedness, left-eyedness and mixed eye and hand dominance are possible but doubtful, and at most slightly influential factors".

One wonders whether in these cases, for some physiological reason, the child is not sufficiently mature at the stage when reading is first introduced, to bear in mind similarities and differences between word sounds and shapes, or to recall with confidence the conventional directional attack. One wonders whether the concept is reasonable, that certain functions underlying any specific learning process, reading included, are slow to mature in certain individuals, to the extent that learning in that area will continue to be impeded for an indefinite period.

Study of the whole child and his problems can suggest lines of treatment; but when help is not fully effective, we find children such as Brian, now almost fourteen years old, enrolled in a course for slow-learners at secondary level. He is known to have decidedly superior ability as measured on a non-verbal test; but his word recognition is very laboured, marked by reversals, poor discrimination of visual pattern, guessing from inadequate data, and the absence of any logical means of attack on unfamiliar words, despite daily exposure to the influences by which other children learn. He is now described as an isolate, disturbed by feelings of inferiority, convinced that he is different from others. Although he has received remedial help over long periods, he has now reached the stage where he makes no effort, experience having shown him that little or nothing is thereby to be gained. One wonders whether in such seriously disabled readers, while remedial measures should be taken to improve reading skill, the emphasis might be removed from book-learning, and these children given more enriched opportunities for developing other skills more in keeping with their assets and strengths. When one realizes that in their anxiety many of the parents of these children curtail their leisure hours, reducing their opportunities for social intercourse or practical achievement in order that they shall spend hour after hour with their books, one wonders whether public opinion could embrace the concept that book-learning is not all important, and that it should be regarded proportionately with other useful and enriching activities.

While it may be said that scientific planning of formal education is still in its early stages of development, much is being done to plan a sequence of learning experiences for young children in direct relationship to individual learning rates and to special learning difficulties. When school experience in the skills of reading, writing, spelling and number work can be tailored for each pupil, it should be possible to reduce learning failure to a minimum, and when failure is inevitable through special handicaps, to minimize unsatisfactory effects. It is in the understanding of the child's strengths and weaknesses that the teaching profession turns to the medical profession for continued guidance and assistance.

## THE MANAGEMENT OF DOUBLE URETERS IN CHILDREN.<sup>1</sup>

By F. D. STEPHENS,  
Melbourne.

CONGENITAL reduplication of the ureter is the anatomical basis of a clinical problem which frequently arises in paediatric practice. At the Royal Children's Hospital, Melbourne, no less than 32 instances of this condition have been studied during the past five years. Hitherto recurrence of infection has all too frequently vitiated the result of carefully planned treatment. I propose to discuss the management of the common types of the abnormality in question, based on a logical understanding of the variants of the condition.

### Types of Double Ureters.

To facilitate description, double ureters may be classified anatomically according to the site of the orifice of the ectopic ureter, into four groups as follows: (i) the external ectopic group, in which the ectopic orifice lies outside the urinary tract in the vulva or lower part of the vagina in the female; (ii) the urethral ectopic group, in which the ectopic orifice opens into the urethra in the female, or into the prostatic urethra or the Wolffian derivatives in the male; (iii) the vesical ectopic group, in which the ectopic orifice opens into the bladder independently; (iv) the conjoined double ureter; in this the two ureters join cranially to the bladder to form a common stem. This conjoined double ureter has been discussed in a previous paper (Stephens, 1956).

For purposes of description in this paper, the ureter draining the lower segment of a double kidney and opening into the bladder in the normal site at the lateral extreme of the trigone is called the orthotopic ureter. This caudal segment of the kidney is termed the orthotopic kidney.

In the external ectopic group, the child is always "wet". The orifice can be found outside the urethral orifice, in the vulva or in the lower part of the vagina.

In the urethral group, the child whose ectopic ureters opens below the internal sphincter zone of the urethra is also "wet". The orifice is invisible from the outside. When the orifice lies in the zone of the internal sphincter, the child is "dry" (Figure 1). There is then an associated ureterocele, which may prolapse; urinary infection is frequent and early.

In the vesical group, urinary infection occurs if one or the other of the ureters permits reflux; but otherwise the group is symptomless.

Conjoined ureters cause short, sharp infections, though if they are enlarged in calibre, the infections may be more prolonged.

### Ureteral Reflux.

In the study of this abnormality, ureteral reflux has been found to occur in one-third of the cases of double ureter. One-third of these ureters with reflux were ectopic, and two-thirds were orthotopic. It is essential, in formulating treatment, to determine which ureters, including the opposite single ureters, are in fact abnormal in this particular.

This is done by the use of micturition cysto-urethrography. During the course of micturition (16% iodide solution is substituted for urine), the high vesical pressure forces urine to the exterior, not only through the urethra, but also into those

<sup>1</sup> Read at a meeting of the Australian Paediatric Association, Canberra, March 30 to April 1, 1957.

ureters in which the defective orifices permit reflux. Reflux, when present, can be clearly demonstrated radiographically.

Reflux from the bladder into the ureters, vesico-ureteral reflux, differs from reflux from the urethra, urethro-ureteral reflux. Vesico-ureteral reflux may occur either when the bladder is filling or during micturition. After micturition the urine flows freely back into the bladder. Urethro-ureteral reflux can occur only during micturition, and the urine involved is locked in the containing ureter until the next act of micturition.

#### *The Management of Reflux by Triple Micturition.*

Reflux in ectopic and orthotopic ureters of the vesical group, and reflux in orthotopic ureters of the urethral group can be managed by triple micturition. Once the initial infection has been overcome, this procedure is necessary only once daily. The residual urine is completely removed when the child performs this exercise effectively. Infection from this source is eliminated by this management.

When reflux is present, the urethral ectopic ureter is filled during micturition, and remains so after micturition though the bladder is empty. The child cannot repeat micturition if the bladder is empty, and triple micturition is therefore not effective in removing the trapped urine from this type of ectopic ureter.

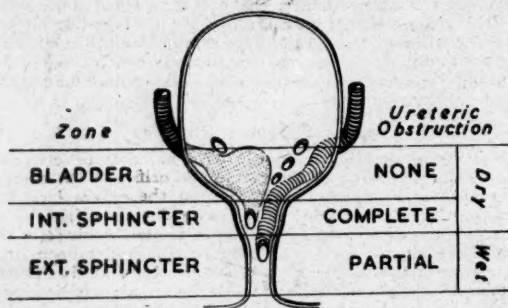


FIGURE 1.

Diagram to show the relationship of the orifice of the ectopic ureter to the sphincter zones of the urethra and to the symptom of "wetting", and to show the obstructive effects of the urethral sphincters on the ectopic ureters.

#### *Special Management of the Urethral Ectopic Ureter.*

The aim of treatment is to eliminate the residual urine which is the cause of infection in this group. This can be done in two ways: (i) by transposing the site of the ureteral orifice from the zone of the obstructing urethral sphincter to the bladder, and then using "multi-micturition" to remove the refluxed urine from the ureter; (ii) by excision of the lower part only of the ureter, or by total ureterectomy and heminephrectomy. The decision as to which course to adopt is determined by the functional value of the ectopic kidney. If it is worth saving, proceed with (i). If it is not worth saving, then proceed with (ii).

#### *Transposition of Urethral Orifice.*

The urethral orifice can be transposed into the bladder by suprapubic or transurethral resection of the roof of the intra-urethral and intravesical ureter or ureteroceles. "Multi-micturition" is performed by the child as for triple micturition, but the efforts to pass urine are continued at two-minute intervals until no more urine comes away. This may mean four or more attempts. Once infection has been overcome, initially with the aid of chemotherapy, multi-micturition is then necessary only once daily; but the procedure must be supervised by the parent.

#### *Excision of the Ureter.*

In the event of the ectopic kidney's being of poor functional value, the management of the ectopic system then depends on the occurrence of reflux into this ureter.

When reflux is absent, the ectopic kidney together with its ureter down to the region of the bladder wall should be removed. The intravesical and intraurethral segments of the ureter then empty and do not refill, and are unlikely to cause any further infection.

When reflux is present, attention is focused on the lower end of the ureter. It is here that reflux occurs, and it is here that the ureteral orifice is blocked off by the contracted sphincters of the urethra.

Excision of the lower end of the ureter as a primary measure is possible only because proximal ligation of the ureter puts the corresponding kidney segment harmlessly out of action. This method of ligation, practised and described by Goldstein and Klotz (1945), has been practised at this hospital by Dr. J. G. Whitaker and myself. We have used this method sometimes in the presence of severe urinary infection, without untoward complications. We advise, however, preliminary uretero-cutaneous drainage prior to ligation when the content of the ureter is thick, creamy pus.

A lower mid-line incision, or a Pfannenstiel incision when the condition is bilateral, gives access to the lower third of the ureter, which can be divided. The ureter is ligated as high as possible.

The lower ureter is then treated by one of the following methods: (a) total excision of the extravascular and submucous intravesical and intraurethral parts of the ureter; (b) extravascular excision and ligation of the ureteral stump at the bladder wall, together with marsupialization of the intravesical and urethral parts of the ureter; (c) extravascular excision together with intravesical and intraurethral excision, care being taken not to interfere with the short intramural tunnel segment, which is ligated outside and inside the bladder. Inside the bladder the tied-off end of the ureter is excluded from the bladder lumen by mucosal approximation with catgut.

All three methods have been used with satisfactory results. In one case in which the ectopic urethral ureter was treated by total excision, the orthotopic ureter, in which previously there had been no reflux, was shown afterwards to permit free reflux necessitating triple micturition. For two other similar ectopic ureters, the same operations were performed without subsequent reflux up the orthotopic ureters. However, operations (b) and (c) would be less likely to induce reflux if, in fact, interference of this intramural segment of the ureters does influence the function of the remaining ureters.

#### *Conclusion.*

Double ureters in children are common, causing symptoms of infection, enuresis, or prolapse of the ureteroceles.

Four types are described according to the site of the orifice of the ectopic ureter—external, urethral, vesical or conjoined.

Children with external and some with urethral ectopic ureters are "wet", while the remainder, though "dry", are particularly prone to infection. Prolapse of the ureteroceles is uncommon.

Ureteral reflux and intermittent ureteral obstruction are the chief causes of residual urine and supervening infection. Urethro-ureteral reflux, when present, occurs only during micturition, with retention in the blocked ureter during the resting and filling stages of vesical function. Vesico-ureteral reflux occurs commonly; but these ureters are not usually obstructed and the refluxed urine can be eliminated by triple micturition. Uretero-ureteral reflux accounts for residual urine and infection in the conjoined types for which heminephrectomy is sometimes indicated.

Treatment is aimed at elimination of the reflux. Surgery is indicated in the external and urethral ectopic groups. The type of surgery varies according to the efficiency of the kidney, the presence of reflux and the degree of obstruction. By the use of ligation of the ureter to put the kidney harmlessly out of action, we have been able to concentrate on the lower end of the ureter with very satisfactory results. This symptomless dormant kidney segment has required no further treatment.

For vesico-ureteral reflux, triple micturition has again proved effective in preventing infections.

In the treatment of double ureters, each ureter must be treated according to its merits, and in any one child different ureters may require different management.

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# PALMAR PULSATION: A PHYSICAL SIGN OF PATENT DUCTUS ARTERIOSUS IN INFANCY.<sup>1</sup>

By DOUGLAS STUCKEY,

Cardiologist, Royal Alexandra Hospital for Children, Sydney.

THE recognition of patent *ductus arteriosus* in infancy offers special problems of its own. In the child who fails to thrive or shows signs of congestive heart failure, this problem becomes a very practical one, because of the possibility of surgical treatment of the condition. Often the murmur is not typical, either in character or in site of maximum intensity, and reliance must be placed on the peripheral arterial signs, such as large amplitude pulses of collapsing quality and a wide pulse pressure. It is often difficult to take accurate blood pressure readings by the cuff method in a small child.

In an infant aged two months, with characteristic signs of patent *ductus arteriosus* later confirmed by operation, it was observed that pulsation of the superficial palmar arch as it crossed the heads of the metacarpals could be easily felt in the palm of the hand as a strong, forcible pulsation. Subsequently, this physical sign was found in a number of children suffering from patent *ductus arteriosus*, and it was decided to try to assess its value from a diagnostic point of view.

## Method and Results.

A group of children in whom the cardio-vascular system was judged to be normal was examined for the presence of palmar pulsation. The results are shown in Table I.

TABLE I.  
Normal Controls.

Age Group. (Years.)	Palmar Pulsation.	
	Absent.	Present.
10 to 12 ..	2	3
8 to 10 ..	7	3
6 to 8 ..	13	3
4 to 6 ..	13	6
2 to 4 ..	17	2
0 to 2 ..	32	2
Total ..	84	18

It will be seen that this sign is uncommon in the age group from birth to two years, and increasingly more common in older age groups. In normal adults these pulsations can usually be found if sought.

A group of children suffering from a variety of congenital heart defects other than patent *ductus arteriosus* were similarly examined, and the results were much the same, as shown in Table II.

In a group of children suffering from patent *ductus arteriosus* subsequently proven at operation, palmar pulsation was almost invariably present, as shown in Table III.

## Mechanism.

Whether an arterial pulsation can be felt or not depends on three main factors. The first is the size and patency of the vessel; the size of a given artery will obviously vary with body size, and the smaller the child, the less likely it is that pulsations will be felt in the more peripheral arteries. Another important factor here is the degree of vasoconstriction or vasodilatation present at the time of examination. The second factor is the accessibility of a particular artery—whether it runs close to the body surface, and whether a hard surface underlies it, making palpation easier. The third factor is the amplitude of the arterial pulsation, which is closely related to the pulse pressure as measured by an intraarterial needle or by the cuff method. It has been shown by Sharpey-Schafer (1953) that peripheral vasodilatation occurs as a baroreceptor response to

increased pulse pressure, so that under these conditions, not only are arterial pulsations of greater amplitude, but they are transmitted further towards the periphery.

The relationship of palmar pulsation to the pulse pressure, as measured by the cuff method in a group of children with a variety of conditions, is shown in Table IV.

TABLE II.  
Congenital Heart Disease (Excluding Patent Ductus Arteriosus).

Age Group. (Years.)	Palmar Pulsation.	
	Absent.	Present.
10 to 12 ..	5	2
8 to 10 ..	5	4
6 to 8 ..	10	2
4 to 6 ..	13	2
2 to 4 ..	18	3
0 to 2 ..	39	1
Totals ..	90	14

TABLE III.  
Patent Ductus Arteriosus.

Age Group. (Years.)	Palmar Pulsation.	
	Absent.	Present.
10 to 12 ..	—	1
8 to 10 ..	—	1
6 to 8 ..	—	2
4 to 6 ..	—	4
2 to 4 ..	—	5
0 to 2 ..	1	8
Total ..	1	21

TABLE IV.

Pulse Pressure. (Millimetres of Mercury.)	Palmar Pulsation.	
	Absent.	Present.
100 or more ..	—	3
95 ..	—	—
90 ..	—	—
85 ..	—	2
80 ..	—	1
75 ..	—	2
70 ..	—	6
65 ..	—	—
60 ..	—	2
55 ..	1	3
50 ..	4	1
45 ..	10	5
40 ..	17	4
35 ..	16	—
30 ..	18	—
25 ..	4	—
20 ..	7	—
Total ..	77	29

It will be seen that if the pulse pressure exceeds 50 millimetres of mercury, this physical sign is almost always present. It is of interest in this regard that in a group of 50 children suffering from patent *ductus arteriosus*, previously reported (Stuckey, 1955), the pulse pressure before operation was greater than 50 millimetres of mercury in the majority, and after operation it was less than 50 millimetres of mercury in all of them.

It must be remembered that any condition which produces increased cardiac output and peripheral vasodilatation is accompanied by a large pulse pressure. The commonest of

<sup>1</sup> Read at a meeting of the Australian Paediatric Association, Canberra, March 30 to April 1, 1957.

such conditions affecting infants are fever, severe anaemia and high environmental temperatures (arbitrarily taken as higher than 80°F.), and any of these factors, either singly or in combination, may produce palmar pulsation in a normal infant. Fortunately, these conditions can usually be avoided or controlled.

#### Conclusions.

1. In the absence of fever, severe anaemia and high environmental temperatures, palmar pulsation is rarely found in normal infants or in infants suffering from various forms of congenital heart disease other than patent *ductus arteriosus*.
2. Palmar pulsation is usually present in children suffering from patent *ductus arteriosus*, and is a useful confirmatory sign of the condition in infants.
3. Absence of palmar pulsation in a child under the age of two years makes it unlikely that a patent *ductus arteriosus* of any size is present.

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### THE PIERRE ROBIN SYNDROME.<sup>1</sup>

By J. STEIGRAD,  
Sydney.

THE apparent frequency of occurrence of troublesome micrognathia in neonates at the Royal Alexandra Hospital for Children led me to attempt a study of our cases, and from this study to evaluate the success or otherwise of surgical treatment of the associated glossoptosis. Reference to our records revealed 10 cases only, all the patients having been admitted to the hospital from 1954 through to the present time. Although it was quite obvious that numerous cases occurred in the preceding years, the cases notes could not be located because of the lack of adequate classification of diagnosis. It was not until July, 1955, that our hospital instituted the present classification, the "Standard Nomenclature of Diseases and Operations", and for the first time was micrognathia included in the classification.

To find the case notes of patients admitted to the hospital during earlier years, search was made under "cleft palate", "congenital deformities" and numerous other categories, but with no success. It became obvious that such of these patients as recovered had been classified under such diagnoses as malnutrition, feeding error or failure to thrive, and those who had died were probably recorded as "pneumonia".

Thus the statistical record of cases of micrognathia at the Royal Alexandra Hospital for Children covers only the past three years, and for this I apologize.

#### Definition.

The syndrome of Pierre Robin, so called after Pierre Robin who wrote about it in 1923, describes infants born with micrognathia and a partial cleft palate. Occasionally other deformities are also found, such as exophthalmos, hypospadias, congenital heart disease, polycystic kidney, etc. The receding lower jaw of these babies is symmetrical, and in profile produces a typical facies, described as "birdface" by some and "Andy Gump" by others.

Anatomically we know that the tongue is normally held forward mainly by the attachment of the genioglossi muscles to the mental spines at the symphysis of the mandible and the frenulum of the tongue at the same site. In micrognathia, the front of the mandible is so far back in its position that the tongue has little if any support (Douglas, 1946). It may therefore fall downward and backward in the lower post-pharyngeal space, covering the epiglottis and acting as a ball valve, egress of air being permitted, but ingress obstructed.

Micrognathia, therefore, becomes important to the paediatrician. The infant presents with a history of episodes

of choking and cyanosis, with marked sternal recession and an inability to swallow adequately. Consequent malnutrition and failure to thrive add to the anxiety of the problem, and excessive salivation plus a tendency to vomit and to aspirate vomitus leads to pneumonia. Death may occur suddenly from suffocation, may result from pneumonia, or may end a long period of semi-starvation.

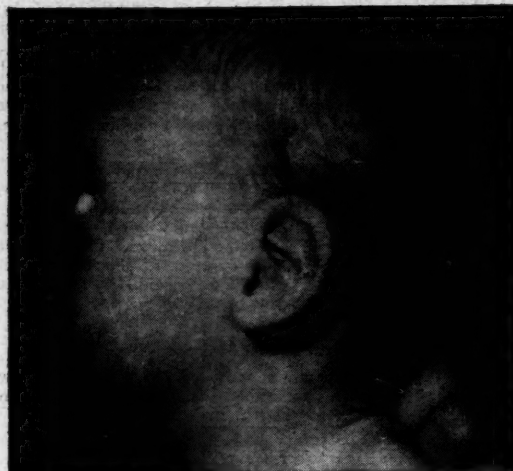


FIGURE I.

#### History.

For many years we have, in a vague way, recognized grades of this condition, and have been alert to the frequent possibility of finding such an infant dead in his cot. It has been realized over many years that constant nursing of the infant prone or on the side was of enormous importance, and of even greater importance was the constant nursing surveillance for twenty-four



FIGURE II.

hours a day. In the same way, a suture passed through the tongue and held to the side of the cot or in some other way to hold the tongue forward was also tried; but only in the past four or five years has the gravity of this syndrome been more clearly understood and a more determined effort made to treat the condition.

<sup>1</sup> Read at a meeting of the Australian Paediatric Association, Canberra, March 30 to April 1, 1957.

Kiskadden and Dietrich (1953) recently reviewed the treatment of micrognathia, and found a report in 1911 by Shukowsky of two cases in which he sutured the tongue to the lower lip. The frequent failure of treatment by positioning stimulated many to try holding the tongue forward by mechanical means. A head brace to force the angles of the jaws forward was devised, tried and given up. Davis and Dunn (1933) introduced an upper-lip guard attached to a nursing bottle, the object of the guard being to force the infant to thrust forward the lower jaw in nursing. Traction by wire through the mandible to a brace and even through a pulley to the framework of the bed was tried. Finally Douglas (1946, 1950) described a technique of sewing the tongue forward to the lower lip in order to create permanent adhesions.

#### Growth of the Mandible.

It is apparent that if the mandible grows forward, the tongue will lose its tendency to fall backwards and obstruct the airway. Pruzansky and Richmond (1955) investigated mandibular growth by means of cephalometric X-ray studies, and found that the lower jaw in infants with micrognathia and cleft palate possessed a remarkable potential for growth. This is confirmed by the fact that children who have had the Pierre Robin syndrome as infants have an almost normal profile at the age of five or six years.

#### Material.

The case notes of 10 patients were studied, and one on whom the Douglas operation had been performed was discarded because the infant suffered from the Treacher Collins syndrome. The remaining nine presented instances of true Pierre Robin syndrome with micrognathia, partial cleft palate and glossoptosis of some degree. Three of the patients died early in the disease, and six recovered, but only after a long period of illness. Three patients were treated by the Douglas operation; none of these died, but one operation only was a total success. A review of the details in the case notes of these patients reveals that the death occurred on the day of admission to hospital and on the third day and on the sixth day after admission in the three fatal cases, and that the infants who recovered spent long periods in hospital for the treatment of their initial symptoms. The average stay in hospital of these patients was 56 days, the shortest being 20 days and the longest 114 days.

It became obvious, in reading the case notes, that in no instance was the referring medical practitioner aware of the syndrome, and that the resident medical staff neither recognized the condition nor realized its potential dangers.

There was no indication in the notes of the nine patients of any heredo-familial basis. However, in one instance, the mother had exophthalmos, as did the patient. In another, the mother was said to have a cleft palate, and to be blind and partially deaf. This infant is also exophthalmic. In still another case the mother had suffered from hydramnios.

#### Conclusions.

1. The syndrome of micrognathia with glossoptosis and consequent cyanosis and sternal recession should receive greater notice.
2. Minor degrees of this condition can be treated adequately by positional nursing; but the infant who is having severe respiratory and feeding difficulties in spite of careful and constant nursing care should be examined by a surgeon, with a view to sewing the tongue to the lower lip according to the method of Douglas.
3. A successful Douglas procedure should provide an adequate airway and permit easier feeding and a rapid weight gain.

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## HEPATITIS IN INFANCY.<sup>1</sup>

By FELIX ARDEN,  
Brisbane.

THIS paper was prompted by the arrival at the Brisbane Children's Hospital in 1955 of three babies with a variety of jaundice which puzzled us at the time, and which has become familiar only during the past few years.

#### Reports of Cases.

CASE I.—The first baby, born prematurely and weighing three pounds, was initially examined at the age of seven weeks, and the early history was vague. By then he had obstructive jaundice, dating, so far as could be told, from birth. On a clinical diagnosis of bile-duct atresia, operation was performed at seventeen weeks, revealing normal biliary passages and free communication with the duodenum. The gall-bladder and ducts contained a clear mucoid fluid, free from bile. The liver appeared healthy to the surgeon, and no biopsy specimen was taken. Three weeks later the stools contained a trace of bile, and over the next two months they regained their proper colour and the jaundice disappeared. The child left hospital at seven months with the liver still slightly enlarged; but at 13 months he weighed 20 pounds and was perfectly well.

CASE II.—The second baby had the distinction of being one of triplets. He also was not examined until the age of seven weeks, by which time the clinical picture of obstructive jaundice was well established. The earlier history was unreliable, but the other two siblings were well. Although a trace of bile pigment appeared in the stools from time to time, the infant was deeply jaundiced, the liver enlarged and the urine bile-stained. A diagnosis of partial biliary tract obstruction had been made, and exploration was being considered, when at the age of eleven weeks spontaneous recovery started, and it was complete in another month.

CASE III.—The third baby, whom I shall describe more fully, was healthy at birth and left the Women's Hospital at the age of seven days, apparently well. Slight jaundice with dark urine appeared at nine days. The motions were said to be of "average" colour, definitely not white. At the age of three weeks, the child arrived in hospital, already 20 ounces above his birth weight, active, alert and with good muscle tone, but moderately jaundiced, with bile in the urine and pale stools. His liver was not enlarged. Investigation revealed a normal blood count, a negative response to the Coombs test, and no maternal antibodies. The serum protein content was 5.6 grammes and the serum bilirubin content 3.6 milligrammes, per 100 millilitres.

For another three weeks the jaundice deepened, the liver enlarged to three fingers' breadth below the costal margin and the stools became colourless. The serum bilirubin level rose to 6 milligrammes per 100 millilitres, cephalin cholesterol flocculation was absent at 24 and 48 hours, and the serum alkaline phosphatase content was 35.4 King-Armstrong units. Tests for syphilis gave negative results. The urine contained bile on every occasion, but reducing substances were absent, and no inclusion bodies were found in centrifuged cells. Urobilinogen was absent from the urine, and various other tests gave negative results. The clinical picture at this stage was one of obstructive jaundice, distinguishable only from atresia of the bile ducts by the history of normally coloured stools for the first days of life.

Needle biopsy of the liver was twice unsuccessfully attempted. After three weeks there came some improvement. The serum bilirubin level fell to 4.2 and then to 3.2 milligrammes per 100 millilitres, the jaundice lessening visibly. Some colour returned to the stools, but the liver remained enlarged. Cephalin cholesterol flocculation, absent at first, appeared slightly (in 48 hours) after six weeks, and more strongly a week later. Meanwhile there was a gradual fading of the jaundice, with disappearance of bile from the urine and its reappearance in the stools. The baby was discharged from hospital after seven weeks. His general condition had been good throughout, with an average weight gain of 10 ounces a week.

#### Comment.

I expect you will agree, even from these brief descriptions, that the three babies, all of whom are now in excellent health, were probably suffering from neonatal hepatitis. The condition is nowadays familiar, but as recently as 1952 it was being described as "inspissated bile of unknown aetiology" (Hsia *et alii*, 1952). Craig and Landing (1952), from a study of the pathological material in the same year, were the first to suggest viral hepatitis.

In the short time remaining, one can only pose and answer a few pertinent questions about this interesting complaint.

How common is neonatal hepatitis? In round figures, 60% of infants with prolonged obstructive jaundice have bile-duct atresia, 15% have obstruction as a post-erythroblastotic state and 25% have neonatal hepatitis. Only 20 cases were collected from 2500 autopsies in 10 years from the Boston Children's Medical Center (Craig and Landing, 1952). There is some evidence that the incidence is increasing.

At what age does the jaundice appear? Many of Harris and Anderson's (1954) 30 patients became jaundiced during the first week. The latest was two months.

What is the pathological evidence that the condition is virus hepatitis? There are now many descriptions in the literature of

<sup>1</sup> Read at a meeting of the Australian Paediatric Association, Canberra, March 30 to April 1, 1957.

biopsy specimens and autopsy material, and these show a striking similarity to the findings in the liver of known hepatitis subjects (either infectious or serum). The major changes are as follows: preservation of the gross architecture of the liver lobule; no regeneration of parenchymatous cells; moderate infiltration of the portal areas; distortion of the liver cell cord pattern due to variation in liver cell size and ballooning of some of the cells; diffuse distribution of multinucleated giant parenchymal cells throughout the lobules; and degenerative changes in the cytoplasm of these cells and to a lesser extent of the other liver cells. The main difference between this pattern and that of adult hepatitis is in the absence of areas of necrosis and subsequent regeneration. In subjects coming to autopsy months after the initial biopsies, progression of the lesions and termination in cirrhosis have been shown.

If the condition is virus hepatitis, is the responsible agent that of infectious hepatitis or that of serum hepatitis? Evidence favours the latter, for the following reasons: (i) The short period of viraemia in cases of infectious hepatitis provides little opportunity for placental transmission. (ii) There has been no history of infectious hepatitis during pregnancy in most cases. (iii) Of 57 mothers who did have infectious hepatitis during pregnancy, only one had a jaundiced baby (Bellin and Bailit, 1952). (iv) The known incubation period of infectious hepatitis infections (14 to 40 days, average 25) excludes post-natal infection, as most subjects are jaundiced within two weeks of birth. (v) In any event, infectious hepatitis is very uncommon under the age of two years. (vi) Prolonged symptomless carriage of serum hepatitis virus is well known. Red Cross experience shows that as many as 0.2% to 0.5% of the general population carry it in their blood. (vii) Evidence is provided by Stokes's experiments. A baby developed neonatal hepatitis at the age of two months. Serum from the mother seven months later produced hepatitis in three of five inmates of a penitentiary at intervals of 75 to 100 days, and serum from the baby likewise in two of five recipients. In due course the infant died of cirrhosis.

Further tests with the mother's serum three years from the confinement date again produced hepatitis in three of five volunteers (Stokes *et alii*, 1954).

Is there a significant family history in these cases? Very little. In Gellis's 41 cases, only one mother had a preceding history of jaundice. None of 25 older siblings was jaundiced, and only one of seven younger siblings (Gellis *et alii*, 1954). Scott, however, reported three affected infants in the same family, with two apparently normal children between the first and second who were affected (Scott, 1954). Tests revealed that the mother herself and both the seemingly healthy children gave a "++++" response to the cephalin cholesterol flocculation test; this finding suggested subclinical infection.

What is the prognosis in neonatal hepatitis? Fairly good. In the largest series, 71% of babies recovered and remained well. Of those who died, some did so as the result of operative interference, some in their first illness with persistent jaundice, and some after a year or two with hepatic cirrhosis (Gellis *et alii*, 1954).

How can the condition be distinguished clinically from atresia of the bile ducts? This can be very difficult. A careful history may confirm the passing of meconium of normal colour and the presence of bile in the stools for the first week or so after birth. Occasionally the obstructive jaundice is not complete. Serum bilirubin levels tend to rise slowly with biliary atresia, and to fluctuate and then fall in hepatitis. Flocculation tests are rather disappointing. Negative results do not rule out hepatitis. Harris and others (1954) found the result of the cephalin cholesterol test positive in only 6 of 25 patients. In their hands, the zinc sulphate turbidity test generally produced a positive result. Diagnostic liver biopsy is justified.

Finally, is exploratory operation warranted? In view of the mortality attending major surgery in these cases, it is best avoided until the child is three or four months old. If it is done, operation may be shortened in the following ways: (i) by taking a cholangiogram with "Diodrast" injected into the gall-bladder; (ii) by examination of a quick-frozen liver biopsy section before the biliary tract is investigated.

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### CONGENITAL DUCTO-PULMONARY ATRESIA: A VARIANT OF PULMONARY ATRESIA OF SOME CLINICAL IMPORTANCE.

By M. L. POWELL, M.R.C.P., F.R.A.C.P.,

AND

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THE title "congenital ducto-pulmonary atresia" is intentionally provocative. Its implications are at present unproven, but it is, we feel, basically correct.

We have, in our records of somewhat more than 1000 congenital heart lesions, 20 cases which we have in the past called *pseudotruncus arteriosus*. It will be remembered that in the true persistent *truncus arteriosus* there is a single large outflow tract from the heart, from which arise the right and left pulmonary arteries as well as the systemic vessels, the innominate, carotid and subclavian arteries. In the so-called "pseudotruncus" there is, in fact, a main pulmonary artery formed separately from the aorta (i.e., the primitive aorta-pulmonary septum has actually formed); but this artery is atretic at some point, and does not actually communicate with the right ventricle.

More peripherally, the right and left pulmonary arteries are patent; but the only practical means of blood supply to the lungs is by grossly enlarged bronchial arteries from the aorta, anastomosing with the pulmonary system in the hilar region. It is this condition, sometimes called "*pseudotruncus arteriosus*" or "pulmonary atresia with large bronchial collaterals", which we would label congenital ducto-pulmonary atresia. The clinical and theoretical aspects of this condition will now be discussed.

#### Clinical Features.

The clinical features are as follows.

Symptoms are relatively mild; the infants thrive quite well, the children have minimal dyspnoea and fair exercise tolerance, and in one case at least, adult life has been reached and maintained in reasonable comfort.

Cyanosis is mild and at times barely noticeable.

Examination of the heart reveals little of specific nature, and the precordium is notably free from bruits in most cases; but, well out in the lung fields, usually maximal in one or sometimes both axillae, is a well-marked continuous bruit. This bruit, be it noted, is not maximum in the ductus area; combined with the cyanosis, it forms a striking diagnostic feature of what we have termed congenital ducto-pulmonary atresia. It is of importance that this bruit is audible at birth.

Blood pressures are normal.

#### Special Investigations.

##### Electrocardiographic Findings.

The electrocardiographic examination shows a right-sided graph with the pattern of right ventricular hypertrophy.

##### Hematological Features.

Investigation of the blood reveals the usual polycythemia and high hemoglobin values of the right-to-left shunts.

##### Radiological Findings.

It is thought that the radiological picture is unique and diagnostic. Various features contribute to the appearance, and the first and most important of these is the increased vascularity

of the lung fields. This in itself is an unusual radiological finding in uncomplicated cyanotic congenital heart disease, once the infant age group is passed. It usually denotes transposition of great vessels or a reversed shunt associated with pulmonary hypertension. However, the congestive changes seen in the lesion under discussion in no way resemble the anatomical architecture of engorged pulmonary arteries, and are in fact due to the enormously enlarged bronchial arteries. The nodular appearance of these vessels is particularly well seen in the upper zones of the lung.

The second important feature is the cardiac contour, which is probably one of the few contours reasonably pathognomonic of the underlying heart lesion. This appearance is produced by a markedly tip-tilted apex denoting a huge hypertrophied right ventricle, an enormous ascending aorta and often a right-sided aortic arch. In the series of 20 patients quoted, 14 showed this right-sided arch, which is a striking phenomenon (Figure 1).

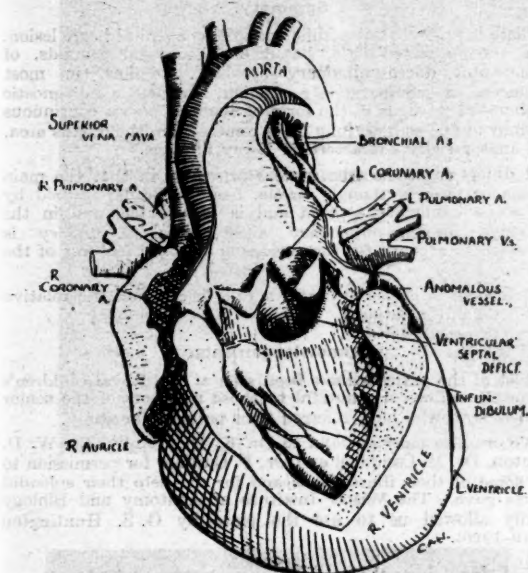


FIGURE IV.

Showing the main pulmonary artery failing to communicate with the right ventricle and the great enlargement of the bronchial vessels anastomosing with the pulmonary system near the lung hilum. An anomalous vessel is also present running from the aorta to the pulmonary artery just above its fused valves. (From Allanby *et alii*, 1950.)

Other important features include the absence of normal pulmonary arteries as shown in the antero-posterior and right anterior oblique views, which is explained by the basic anatomy of the lesion. The use of a barium bolus may also demonstrate indentations of the esophagus due to the huge bronchial arteries (Figure II).

#### Angiocardiographic Findings.

Eleven of the patients at the Royal Children's Hospital have had an angiocardiographic examination and in all cases this has been typical. The huge aorta overlying both ventricles has filled at once from the right ventricle, and in most cases the bronchial arteries can be seen arising from this. Often an insignificant right ventricular outflow tract or infundibulum dwindling away to nothing can be seen, especially in the right anterior oblique position (Figure III). Special mention must be made of the size of the ascending aorta, the diameter of which is often three times that of the descending aorta (see Figure III).

#### Cardiac Catheterization.

It is thought that angiocardiography in this condition is a more useful investigation than cardiac catheterization. In the one case in which a catheter was passed, the oxygen tension showed a rise, high in the right ventricle, and the right ventricular pressure was equal to the systemic pressure.

#### Differential Diagnosis.

The differential diagnosis is from an arterio-venous aneurysm of the lung, which is eliminated by X-ray findings, or a tetrad of Fallot with an open ductus arteriosus; but here the continuous murmur is in the ductus area, and the radiological features are different. It should be noted that in one of our cases the continuous murmur actually was loudest in approximately the ductus area; it is possible that an anomalous vessel was present as in one of the cases of Allanby *et alii* (1950) (Figure IV).

#### Pathological Anatomy.

The basic abnormality is pulmonary artery atresia, due, it is thought, to grossly unequal division of the primitive *bulbus cordis* by the aorto-pulmonary septum. This atresia does not necessarily extend throughout the two main branches of the pulmonary artery, but certainly is present at its origin, thus precluding its junction with the right ventricle. Under this condition two other possible channels remain for supply to the lung fields—the bronchial arteries and the ductus arteriosus.

In this lesion it is the bronchial arteries which enlarge to massive proportions, enter into communications with the pulmonary system near the hilum of the lung, and thereby produce the arterio-venous shunts and curiously placed continuous murmurs which are the hallmark of the condition.

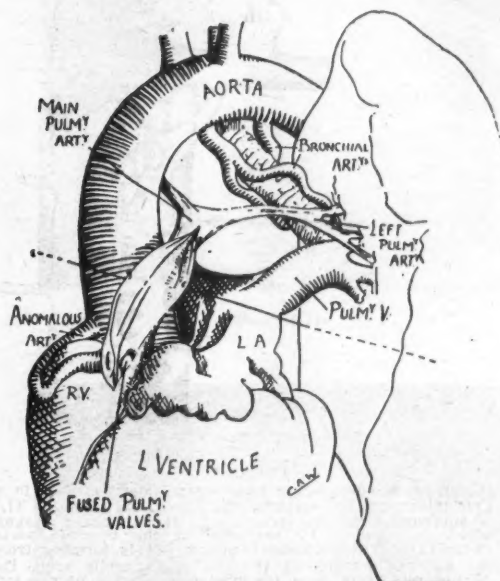


FIGURE V.

Showing the interior of the heart from the right side. The aorta is large and is the sole outflow tract. It overrides both ventricles, and a large ventricular septal defect is present. The pulmonary artery does not make contact with the right ventricle. (From Allanby *et alii*, 1950.)

Normally, the bronchial arteries, usually three in number, arise from the upper part of the descending aorta; occasionally one may come from the first or second intercostal artery. They supply the bronchi and extend as far as the alveolar ducts, the return being mainly via the pulmonary veins. No large anastomoses occur with the pulmonary system except under exceptional circumstances, such as the condition under discussion.

Sometimes other vessels enter the picture, as in a case of Allanby *et alii* (1950) (Figure V), in which an abnormal artery left the aortic sinus close to the left coronary artery and entered the pulmonary artery above the fused valves.

The aorta, which is the sole outflow tract from the heart, is huge, and overrides the interventricular septum (Figure V).

No pulmonary arteries leave the aorta, as in the true *truncus arteriosus*, a lesion of great rarity, in which the primitive aorto-pulmonary septum has never appeared. The aorta has been right-sided in a high proportion of our cases. The right ventricle

is grossly hypertrophied, for it is sharing with the left ventricle the total cardiac output.

The role of the *ductus arteriosus* in this lesion is of interest. It is suggested that the reason why the bronchial arteries, which never normally anastomose with the pulmonary arterial system, are so large and freely communicable with this septum, even at birth, is that, in addition to pulmonary atresia, there is also an atresic, non-functioning, or absent *ductus arteriosus* throughout intrauterine life. As can be seen from the cat embryo models of Huntington (1919) (Figure VI), this must delegate to the bronchial



FIGURE VI.

Model of the developing pulmonary artery system in a four millimetre cat embryo. The pulmonary Anlage (L) is surrounded by the post-bronchial pulmonary plexus, which is supplied by branches of the thoracic aorta (8 to 14). The pulmonary artery (6) is forming from the anterior portion of the left sixth aortic arch, the *ductus arteriosus* from the posterior portion of the left sixth aortic arch. (In the cat the *ductus arteriosus* is bifid and includes also a branch from the dorsal aorta—7.) It may be seen from this diagram that should the whole of the left sixth arch fail to develop fully—i.e., the pulmonary artery and the *ductus arteriosus* be deficient—there is only one possible supply to the lungs, and that is the branches from the dorsal aorta—i.e., the bronchial arteries. These will necessarily be large at birth. (From Huntington, 1919-1920.)

arteries the sole supply to the developing lungs. The intimate relations of the pulmonary and bronchial supply in the primitive pulmonary plexus explain the ready establishment of the large shunts so characteristic of this condition. In the tetrad of Fallot such shunts would be advantageous; but the presence of a normal *ductus arteriosus* prevents this development, and in the tetrad it is not for some years after birth that the bronchial arteries enlarge appreciably.

This theory is not easy to prove, for it is difficult to say whether a *ligamentum arteriosum* has ever been a functioning *ductus arteriosus*; but a series of cases reported by Allanby *et alii* (1950) certainly does not disprove it. In fact, one post-mortem examination was reported as revealing a very large bronchial-pulmonary artery shunt; the *ductus* was not mentioned at all, and no sign of a ligament can be seen in the illustration of the lesion.

#### Post-Mortem Material.

So well have these infants and children been, that we have been denied the important information afforded by post-mortem material. This, of course, is a criticism of the suggested title for the lesion.

#### Treatment.

Treatment has been conservative in all our cases, as the children are relatively so well. If deterioration occurs, it may be possible to increase the flow to the lungs by a Blalock anastomosis. If so, this must be performed on the side opposite to the loudest continuous murmur, for that is the side receiving maximum bronchial flow and collapse of that lung for operative purposes could be fatal (Roche, 1953). One would think, however, that if the enlarged bronchial arteries were unequal to the task of supplying the lung fields, it would be unlikely that surgery would achieve worthwhile improvement.

#### Summary.

There is presented a specific cyanotic congenital heart lesion, with the suggested title, based on theoretical grounds, of "congenital ducto-pulmonary atresia". It has, in most instances, a comparatively benign course, a diagnostic combination of signs in the form of cyanosis plus a continuous murmur in one or both lung fields remote from the ductus area, and anatomically characteristic X-ray findings.

It differs from the true *truncus arteriosus* in that the main pulmonary artery, though atresic, has been differentiated by the aorto-pulmonary septum and is identifiable, and in the extensive broncho-pulmonary anastomoses. A theory is advanced to account for the curious anatomical features of the lesion.

Treatment is conservative as a rule, because of the relative well-being of the patients.

#### Acknowledgements.

Most of the patients have been seen at the Royal Children's Hospital, and we are grateful to those members of the senior medical staff who have referred their patients for study.

We are also most grateful to Dr. K. D. Allanby, Dr. W. D. Brinton, Dr. M. Campbell and Dr. F. Gardner for permission to use some of their illustrations and for the help their splendid article gave. The Wistar Institute of Anatomy and Biology kindly allowed us to use the plate by G. S. Huntington (1919-1920).

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### Reports of Cases.

#### ACUTE HÆMOLYTIC ANÆMIA ASSOCIATED WITH ACUTE GLOMERULONEPHRITIS IN INFANCY.<sup>1</sup>

By S. E. J. ROBERTSON,  
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This short paper is to draw attention to the association of acute hæmolytic anæmia with acute glomerulonephritis as seen in two infants in the last year.

#### Clinical Record.

Both the patients were under the age of 12 months, one being aged five months and the other ten months. The clinical features in each case were essentially the same, consisting of an upper respiratory tract illness followed about two weeks later by anorexia, irritability, vomiting, the passage of abnormal stools and dark urine, and rapidly increasing pallor. Both infants

<sup>1</sup> Read at a meeting of the Australian Pediatric Association, Canberra, March 30 to April 1, 1957.

ILLUSTRATIONS TO THE ARTICLE BY J. M. ALEXANDER, M.B., B.S., M.R.A.C.P.

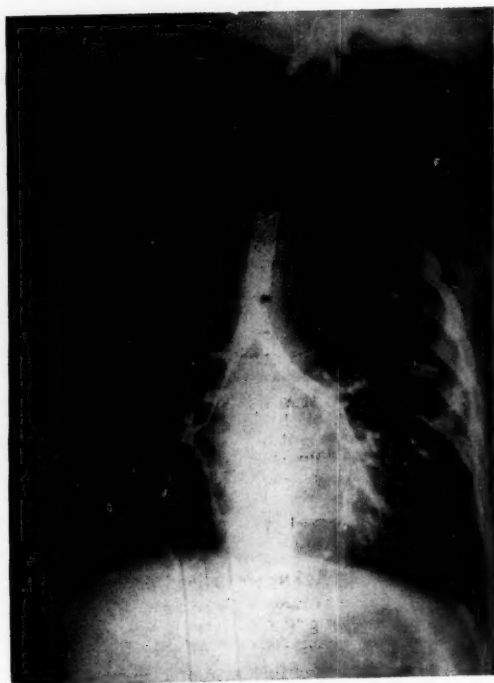


FIGURE I.

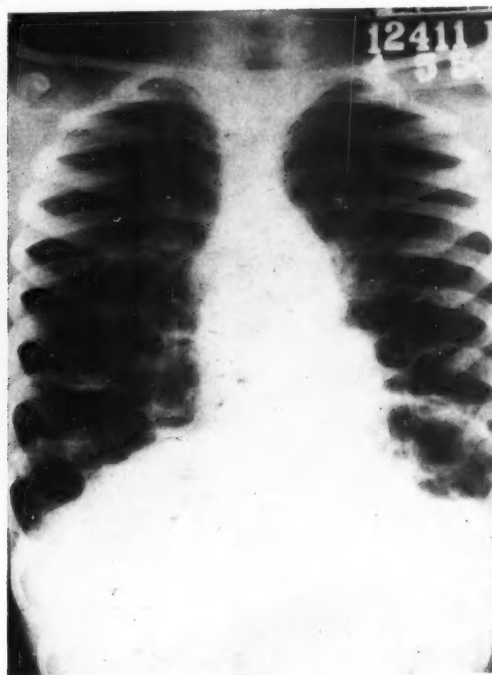


FIGURE II.



FIGURE III.

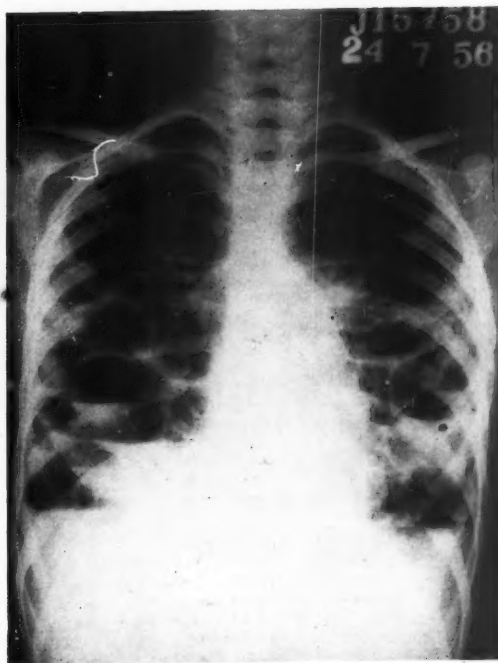
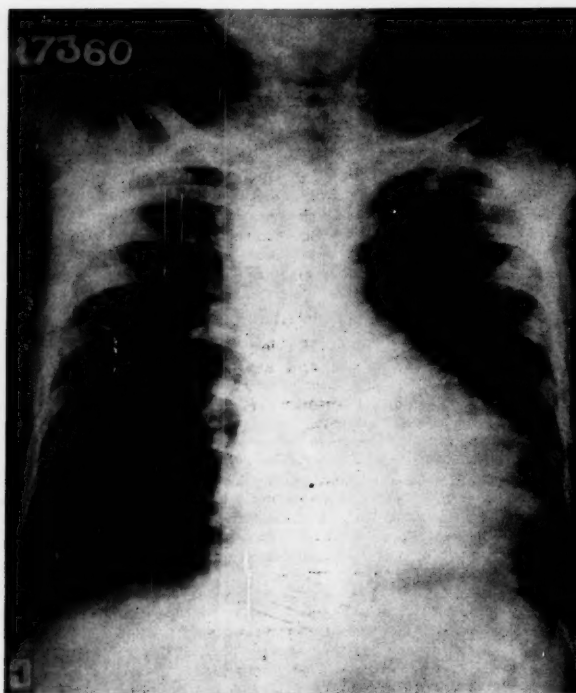


FIGURE IV.

ILLUSTRATIONS TO THE ARTICLE BY M. L. POWELL, M.R.C.P., F.R.A.C.P., AND H. G. HILLER, M.D., M.R.A.C.P.,  
D.D.R., M.R.C.A.



were admitted to hospital within a few days of the onset of these symptoms, one with the provisional diagnosis of intestinal obstruction and the other as probably suffering from gastro-enteritis.

When examined on their admission, the infants were fretful and very pale, with mild fever and tachycardia. One infant was slightly jaundiced. No oedema was detected, and there was no clinical enlargement of the liver or spleen. The upper parts of the respiratory tracts and the lungs appeared normal. No abnormality could be detected on examination of the heart, but the blood pressure could be considered slightly elevated; the figures were 100/70 millimetres of mercury in one case, and 105/75 in the other.

Examination of the blood revealed anaemia and a neutrophil leucocytosis. The anaemia was considered to be hemolytic because of an accompanying increase in nucleated red cells and reticulocytes in the peripheral blood, an increase in the serum bilirubin level and marked normoblastic hyperplasia of the bone marrow in both patients. The figures are shown in Table I.

TABLE I.

Observation.	Case I.	Case II.
Red cells per cubic millimetre	3,100,000	1,740,000
Hæmoglobin value (grammes per centum)	7.6	4.7
Reticulocytes (percentage of red cells)	3.5	2.5
Nucleated red cells per cubic millimetre	2000	19,000
Leucocytes per cubic millimetre	14,000	44,000
Neutrophils per cubic millimetre	7000	20,000
Serum bilirubin content (milligrammes per 100 millilitres)	2.2	2.5

Examination of stained films of the peripheral blood showed, apart from the increase in nucleated red cells, anisocytosis, macrocytosis, polychromasia and some small darkly-stained cells suggestive of spherocytes. The direct Coombs test was carried out on several occasions in each case but gave consistently negative results. No autoimmune antibodies could be detected in the serum. The fragility of incubated red cells in both cases was slightly increased.

No relatives of either patient had anaemia, jaundice, splenomegaly or gall-bladder disease. Examination of a stained smear of the peripheral blood of both parents of each child showed no evidence of microspherocytosis.

Examination of urine collected from each patient showed it to be of normal colour; but further examination revealed "heavy" albuminuria, with numerous red cells and casts of the hyaline and granular variety. No hæmoglobin, methæmoglobin or urobilinogen was found. The blood urea level in the first patient was 47 milligrammes, and in the second 70 milligrammes, per 100 millilitres.

An interesting feature was the distinct orange colour of the stools, which were surrounded on the napkin by a pink stain. This pink stain was found to be due to urobilinogen.

The most obvious disturbance in both these patients was the rapidly developing anaemia. This appeared to be due to hemolysis, in view of the distinct evidence of rapid red-cell regeneration, as shown by the increase in circulating nucleated red cells, the reticulocytosis and the normoblastic hyperplasia of the bone marrow, plus the evidence of red-cell destruction, as shown by the increased level of serum bilirubin and the large amount of urobilinogen in the faeces. Accompanying this anaemia was acute glomerulonephritis, as shown by albuminuria, hæmaturia and cylinduria, with a rise in the blood urea level and mild hypertension.

Subsequent progress was essentially the same in both cases. Anorexia, fever, vomiting and the passage of loose, orange-coloured stools continued. The hæmoglobin value of the first patient fell from 7.6 to 5.6 grammes per centum in three days. Transfusions of the red cells from 200 cubic centimetres of blood were given to each patient as soon as the investigations necessary for diagnosis had been completed. These transfusions raised the hæmoglobin levels to normal, and this was accompanied by restoration of a normal skin colour, appetite and vigour, with cessation of vomiting. However, after a period of three days, pallor, anorexia, irritability and vomiting recurred with a sharp fall in the hæmoglobin levels. The first patient was again given the red cells from 200 cubic centimetres of blood

and the second those from 500 cubic centimetres. The first patient again developed pallor and anorexia after several days and required a third and final transfusion. During these relapses, no change occurred in the urinary findings, but slight enlargement of the spleen was noticed. The hæmoglobin values were subsequently estimated at regular intervals for a year in the first case and for six months in the second, and no fall in the levels below normal was observed.

Neither during nor subsequent to the acute phase of the illness was any source of bleeding discovered, and the hæmaturia was always microscopically evident only. Despite the evidence of mild spherocytosis and increased fragility, which is common in acute acquired hemolytic anaemia, congenital hemolytic anaemia could be ruled out by the rapid relapse in both cases after the initial transfusion, by the absence of any suggestive family history and by the subsequent normal course of each infant.

The red cells and casts in the urine diminished to nil over a period of three weeks, and the albuminuria became only a trace. During this period the blood pressures and the blood urea levels fell to normal. Six months after the initial illnesses, no abnormality could be detected in the urine. The subsequent health, activity and growth of both children have been normal.

### Discussion.

Attention was drawn by Hensley (1952) to the association of acute hemolytic anaemia and acute glomerulonephritis. He described three patients, two of whom were infants, with acute hemolytic anaemia, with autopsy findings confirming the presence of acute glomerulonephritis. It has long been known that anaemia of varying degrees of severity usually accompanies acute glomerulonephritis. Burke and Ross (1947) analysed 90 consecutive cases of acute glomerulonephritis in children over a three-year period. He found hæmoglobin levels of 8 grammes per centum or less in 20%, and a level between 9 and 10 grammes per centum in 52%. He did not mention the ages of the children with regard to these hæmoglobin levels. Acute glomerulonephritis in small infants appears to be uncommon, but when it does occur, anaemia appears to be particularly severe. Search of the records at the Royal Alexandra Hospital for Children for the period of the last two years could produce only two infants with acute glomerulonephritis under the age of 12 months, apart from the two described in this paper. The hæmoglobin level was 5.6 grammes per centum in one and 7.0 grammes per centum in the other. No extensive investigations of the anaemia were carried out, but it was noticeable that nucleated red cells were frequent in the stained films of the peripheral blood, and that the direct Coombs test produced a negative result in the one case in which this test was performed. Fison (1956) reviewed the subject of acute glomerulonephritis in infancy. He did not emphasize the anaemia; but in the two fatal cases he reported, the hæmoglobin levels were 4.9 and 6.2 grammes per centum respectively. No detailed study of the anaemia was made, but nucleated red cells were prominent in the peripheral blood, and the result of the direct Coombs test was negative in the one patient in whom this test was carried out.

Very little is known of the cause of this anaemia which accompanies acute nephritis, and most of the literature on this point deals with chronic renal disease. It has long been thought that some, as yet undemonstrable, toxin depresses bone-marrow function (Parsons and Ekola-Strolberg, 1933); but no cellular deficiency of the bone marrow is usually demonstrable, and in many cases red-cell hyperplasia is present. Emerson (1949) showed that there was diminished red-cell survival time in acute glomerulonephritis, and most authorities now agree that increased hemolysis plays a major part in the causation of this anaemia.

On the other hand, acute acquired hemolytic anaemia due to a circulating isohemolysin is not an uncommon disease in small infants. In such patients evidence of renal inflammation is lacking, and there is often a positive response to the direct Coombs test, indicating sensitization of the red cells by an antigen-antibody reaction. Acute glomerulonephritis, present in the cases described in this paper, is thought to be due to an antigen-antibody reaction affecting the renal tissues, and it is reasonable to suggest that accompanying acute hemolytic anaemia is due to sensitization of the red cells in a similar way. The fact that a positive response to the direct Coombs test was not demonstrable in the cases in this paper, in the cases of acute glomerulonephritis and anaemia in the literature or in the

records of the Royal Alexandra Hospital for Children may indicate that the sensitization of the red cells is of a different type from that which occurs in the better-known acute acquired hemolytic anemia, in which there is no evidence of renal inflammation. The association of acute glomerulonephritis and acute hemolytic anemia in the cases described suggests that study of the anemia of acute glomerulonephritis, especially in infancy, may show that this anemia is largely, if not wholly, hemolytic in origin.

#### Summary.

Two cases of acute glomerulonephritis and acute hemolytic anemia in patients aged under 12 months have been described. Both patients recovered completely after treatment with repeated blood transfusions. From a study of these cases and of similar cases in the literature, it is suggested that the anemia of acute glomerulonephritis is hemolytic in origin.

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### ECTOPIC URETER.<sup>1</sup>

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ECTOPIC URETER is a condition in which there has been a duplication of the primitive ureteric bud in an early stage of embryonic development. Two ureters develop from this bud, and the kidney therefore has two separate pelves. The condition may be bilateral, but is usually unilateral. On the affected side there may be two ureteric openings within the bladder; but more often one of the ureters opens into the urethra, or into the vaginal vestibule without a bladder opening.

In the male this abnormal opening is in the prostatic part of the urethra, or even in the ejaculatory ducts, and is always above the voluntary sphincter urethrae; consequently it is rare for the affected male to be incontinent of urine. In the female the opening is often beyond the less well-developed voluntary sphincter, and involuntary leakage of urine is common.

It is nearly always the ureter from the upper pelvis of the double kidney which opens in this ectopic fashion, owing to a curious taking up of the lower part of the Wolffian duct in the bladder wall during development. It usually happens that the ectopic ureter which drains the upper pole of the kidney develops gross dilatation and tortuosity except at its lower end, and the upper pole of the affected kidney is often small and fibrotic from back pressure and infection. It may even cease to function. Usually the rest of the kidney functions well, and the normally situated ureter is healthy; but this is not always so, since this ureter may be partly blocked by the abnormal ureter, either where the two cross in the pelvis, or owing to the protrusion caused in the trigonal region by the dilated ectopic ureter—a type of ureterocele.

The characteristic symptoms, apart from complicating infections, are a type of false incontinence of urine, usually in a female child. The so-called incontinence takes the form of a fairly constant, though slight, leakage of urine, especially in the upright position. This leakage is quite beyond the power of the child to control; but it is found, on careful questioning, that the child can and does empty the bladder at intervals in a perfectly normal manner.

Such a story is pathognomonic; but the story may not be so definite, and many cases are not diagnosed, even in adult life, the result being prolonged misery and social ostracism. The

condition, if recognized, is readily cured in uncomplicated cases by heminephrectomy with removal of the abnormal ureter.

It is my intention to present to you details of three patients with ectopic ureter who have come into my beds at the Royal Alexandra Hospital for Children, Sydney, during the past three years. They form an interesting group, because each presented in an entirely different way, and each presented his own peculiar problem in investigation and diagnosis. These three stories should make us realize that congenital anomalies of ureteric development are not infrequent, and may account for quite bizarre clinical pictures, which can be elucidated only if the physician is alert to the possibility of such abnormality and willing to submit the child to full urinary tract investigation. I should like to emphasize at this point that even a so-called normal excretory pyelogram does not exclude ectopic ureter, because frequently the affected upper pole is small, and there is failure to show the second ureter because of poor concentrating power in the affected upper pole.

#### Case I.

A female child was nine years old when Dr. K. Winning asked me to examine her on September 21, 1953. She had a typical history of persistent slight urinary dribbling since infancy. Despite this constant leakage, she emptied her bladder normally at normal intervals. While lying in bed in hospital she remained "dry". Her urine was not infected and her blood urea level was normal. Her excretory pyelogram was reported as showing normal appearances on both sides; but subsequent reexamination suggested a missing upper calyceal system on the left side.

On close examination of the child under anesthesia, the abnormal ureteric opening was found in the vulva just posterolateral to the normal meatus, and a ureteric catheter could be passed into it. A retrograde pyelogram of this ureter was obtained, in addition to pyelograms after cystoscopy and catheterization of both normal ureters. The X-ray films showed clearly that the ectopic ureter led to the upper pole of the left kidney, and that this ureter and the pelvis were dilated.

On September 21, the left kidney was exposed, the two kidney pelves and ureters being revealed. The upper third of the kidney and its ureter were removed as far as the pelvic floor. Convalescence was rapid and satisfactory, the child no longer had any leakage of urine, and a post-operative excretory pyelogram showed satisfactory appearances.

#### Comment.

This was a very satisfactory ending to a long story, and was really a typical case, in which diagnosis should have been made earlier.

#### Case II.

The second patient, a female, was only three months old when admitted to hospital on August 25, 1953, under the care of Dr. Winning, with the diagnosis of gastro-enteritis. Four days before her admission she had begun to pass loose stools (six to eight per day), which had persisted despite treatment with diet restriction and "Aureomycin". Two days after her admission it was discovered that she had a mass with tenderness and guarding in the right iliac fossa, and in the pelvis. She had a leucocyte count of 39,000 per cubic millimetre. Despite her tender age, a diagnosis of appendiceal abscess was made. Operation was performed by Dr. F. N. Street, and disclosed a normal appendix, Fallopian tube and ovary; but there was a retroperitoneal mass. The peritoneum was closed and the mass exposed. It appeared to extend up and down in the line of the ureter, and when an opening was made into it, thick, creamy pus was obtained in large amount. A drainage tube was inserted and the wound was closed. She was a very sick little baby for three or four days and needed intravenous maintenance. Later she settled down.

On September 15, subcutaneous pyelography was carried out, and the report was that dye was excreted promptly on both sides, and the renal pelves appeared normal. This was a great surprise, as I had expected to find a functionless right kidney. The urine was normal. It was at this stage that we first suspected an ectopic ureter.

A second pyelographic examination was made on September 29, with no change in the appearances; however, the radiologist suggested that the right pelvic calyceal system seemed to correspond in size with the lower and middle calyceal groups of the left kidney. The baby was sent home. She remained perfectly

<sup>1</sup> Read at a meeting of the Australian Paediatric Association, Canberra, March 30 to April 1, 1957.

well, and was readmitted to hospital for investigation when twelve months old. Her urine was normal, her blood urea level was 40 milligrammes per 100 millilitres, her hemoglobin value was 13.8 milligrammes per 100 millilitres, and her erythrocyte sedimentation rate was four millimetres in one hour. Subcutaneous pyelography again revealed no abnormality although the shadow on the right showed the same somewhat rudimentary appearance. A confident diagnosis of ectopic ureter was made, but it was decided not to treat this till the patient was older.

On October 4, 1955, at the age of two years and three months, she was readmitted to hospital. She still had a tiny sinus on the abdominal wall, which occasionally discharged clear yellow fluid *plus* mucus in small quantities. She had been quite well, and had no incontinence of urine. Under anaesthesia, cystoscopic examination revealed no abnormality in the bladder; but close examination of the vulva revealed that she had a partly septate vagina at its lower end. No abnormal ureteric opening could be seen. The sinus on the abdominal wall was dilated, and then catheterized and filled with "Lipiodol". X-ray examination showed that the sinus led to the region of the right kidney, and drained a tiny shrunken upper calyx. Once again, we sent her home.

On May 24, 1956, when the child was three years old and quite healthy, the right kidney was exposed and a tiny shrunken upper pole was removed with its ureter, which ran to the region of the sinus, and beyond that again down into the pelvis, where it was excised. There were no post-operative difficulties.

Dr. R. D. K. Reye reported later that examination of the specimen revealed a small sclerosed segment of renal tissue, and then a band of fibrous tissue, with a fringe of normal kidney beyond it. In the ureter, chronic inflammatory cells were present in cedematous mucosa.

#### Comment.

This child remains a healthy little girl, but has recently had a urinary infection, presumably due to trouble with the residual lower end of her ectopic ureter, and will need further investigation. The story as it evolved has been a fascinating one.

#### Case III.

The third patient, a female, was also admitted to hospital in the care of Dr. Winning, on May 17, 1955, at the age of ten weeks. She had failed to thrive, and had had intermittent vomiting and fever beginning at the age of two weeks. At the age of seven weeks she had been admitted to Tresillian Mothercraft Home, still weighing 7 pounds 9 ounces—the same as at birth. There she had been found to have a urinary infection, which cleared up with sulphonamide treatment, but recurred as soon as this was stopped. A small cystic protrusion had been noted at the vulva. Her blood urea level was 24 milligrammes per 100 millilitres. On May 25, subcutaneous pyelography was carried out; up to two hours there was no excretion from the left kidney, but the right kidney was normal. At three hours a left-sided hydronephrosis with tortuous dilated ureter was seen, probably due to reflux of dye from the bladder. The radiologist had not commented on a rather obvious filling defect in the bladder shadow.

It was at this stage that I was asked to examine her. The filling defect was noted, and the renal tract dilatation was thought to be due to the cyst. On May 30, examination of the child under anaesthesia showed that the cyst was in the anterior vaginal wall proximal to the normal urethral opening. The cyst was converted to a cave by removing portion of its roof. The pathologist reported that the tissue removed was lined on one side by vaginal epithelium, but on the other by transitional epithelium of bladder type.

The baby continued to have persistent urinary infection but gained weight well. Excretory pyelographic examination on June 7 showed well marked left-sided hydronephrosis at two hours, but less marked than before. On July 5, there was no appreciable change in the pyelographic picture. On the following day, under anaesthesia, cystoscopic examination revealed a normal vagina and cervix, and normal bladder mucosa except for some trigonitis. The right ureteric orifice seemed normal, the left could not be seen. No ureterocele was seen. The child was allowed home, apparently well.

A month later she was readmitted to hospital for further pyelographic examination. The urine was still infected, and her mother also described the passage on occasions of thick pieces

of debris, which she considered came from the vagina. There was no change in the excretory pyelogram, and it was considered that eventually nephrectomy would need to be performed. On September 20, she was readmitted to hospital at the age of six months. She had been fairly well, but still had an intermittent "vaginal" discharge. On examination of the child under anaesthesia, the urethral opening was rather high in the vaginal wall, and sounds passed as readily into the "cave" left after the unroofing of the cyst as into the bladder. Pus exuded from the former. Cystoscopic examination revealed a soft cystic protrusion in the trigonal area, which almost obscured the ureteric openings, especially the left; this was seen to be patulous, and there were flakes of fibrin or pus in the urine which came from it. Both ureters were catheterized, and the pyelograms obtained confirmed the left hydronephrosis and suggested that the left kidney pelvis was incomplete.

It was considered at this stage that there must be an ectopic ureter from the upper pole of the left kidney, that its lower end was the cause of the ureterocele seen in the trigone and that it had also been the cause of the original vulval cyst.

On October 5, a cystographic examination was carried out, the catheter being guided with some difficulty into the bladder rather than the "cave". There was free reflux to the left kidney from a left ureter which seemed to arise from a sort of diverticulum of the bladder, the right side of the bladder floor being elevated by the ureterocele or "cyst".

At the age of fourteen months the baby was readmitted to hospital for further investigation. She was a healthy baby of normal weight and was walking. Her mother said that she still passed pus in "ropes" at times. Her blood urea content was 40 milligrammes per 100 millilitres, and a mild urinary infection was still present. On May 17, 1956, two openings were again noted close together in the anterior vaginal wall, and the abnormal cul-de-sac admitted a sound about three inches beneath the bladder base. Dye was injected into this cul-de-sac, but ran back freely into the vagina. Nevertheless, X-ray examination showed some dye running up the ectopic ureter to the upper pole of the left kidney. The hydroureter leading to the main kidney pelvis was not filled.

An excretory pyelogram showed no excretion from the left side at two and a half hours or at three and a half hours. Accordingly nephrectomy was carried out on May 28, and a hydronephrotic kidney with two pelves and two ureters was removed. The upper ureter crossed the lower one in the pelvis and appeared to be obstructing it at this point. The upper pole was shrunken and fibrous.

The renal tissue related to the ectopic ureter consisted of two tough-walled cysts with smooth inner linings. Microscopic examination of the functional segment of the kidney showed slightly compressed parenchyma with lymphocytic infiltration of the pelvic mucosa, but none in the cortex or medulla.

#### Comment.

The child has remained well since operation, and it is considered that it may not be necessary to do anything further to the remaining lower segment of the ectopic ureter.

### A DIENCEPHALIC SYNDROME OF EARLY INFANCY.<sup>1</sup>

By LORIMER DODS,

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In October, 1955, a severely emaciated male infant, A., aged seven months, was referred to the clinical unit of The Institute of Child Health by Dr. Kathleen Winning because of irregular but persistent vomiting, which had commenced at the age of about two months. A long series of clinical, radiological and laboratory investigations had failed to establish any explanation for this child's vomiting or for his severe degree of emaciation, which was obviously out of proportion to his caloric loss through vomiting. In spite of his extreme wasting, several months spent in hospital wards and a lengthy programme of investigations, he was surprisingly happy, almost "defiantly gay", amazingly alert

<sup>1</sup> Read at a meeting of the Australian Paediatric Association, Canberra, March 30 to April 1, 1957.

and vigorous and apparently full of affection for everybody, even including those people in long white coats who took blood from his veins. During his stay in hospital, three new clinical manifestations (Figure 1) became apparent: first, his obvious and constant pallor, despite the absence of any significant anaemia; secondly, his unceasing hyperkinesia—continual, apparently purposeless, "weaving" movements of his limbs, which were not diminished by generous doses of sedative and "tranquillizing" drugs, and which were present to some degree during sleep; thirdly, his excessive sweating, which was generalized, more marked during feeding and evident even in the winter months. Although comments by the nursing staff suggested the possibility of polyuria, this suspicion was never confirmed by accurate measurements of his urinary output.

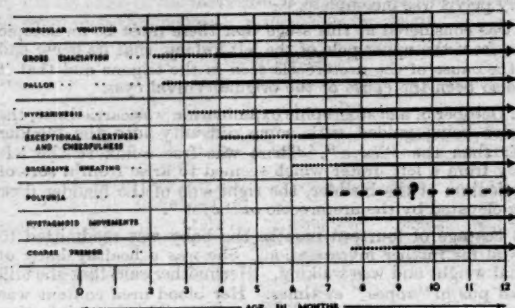


FIGURE 1.

Clinical features: patient A. Final diagnosis, oligodendroglioma invading the third ventricle.

This combination of emaciation, pallor, hyperkinesia, exceptional alertness and vigour, excessive sweating, a slender suggestion of something akin to euphoria and some variable nystagmoid movements (noted at a later stage of his illness) suggested the possibility of a diencephalic lesion. It was thought that the clinical picture corresponded to some degree with the diencephalic syndrome very briefly described by Russell (1951), and this similarity prompted a letter to Russell. Russell's very helpful reply gave some support to the tentative diagnosis of a diencephalic lesion, despite the absence in this particular case of a number of clinical features stressed by him, such as tachycardia, vascular hypertension or hypotension,

hypoglycaemia and a striking discrepancy between the infant's falling weight and his rapidly advancing height (Russell, 1956).

During a period of seven months' observation, a single examination of this infant's cerebro-spinal fluid and repeated examinations of his fundi gave negative results, and two radiographs of his skull did not reveal any signs suggestive of increased intracranial pressure. At the age of 19 months he died in another hospital, and a post-mortem examination disclosed an oligodendroglioma of the optic chiasma which had invaded and filled the third ventricle and also—as a special clinical humiliation to me—"congenital hypertrophic pyloric stenosis", a finding which may raise in some minds certain hypothetical considerations about the possible part played by the autonomic system in the aetiology of this disorder of the pylorus.

Less than a year later, another male infant B., was admitted to the Royal Alexandra Hospital for Children at the age of five months because of progressive and unexplained emaciation associated with irregular vomiting, which had begun at the age of about three months (Table I). This grossly emaciated infant was surprisingly happy and amazingly vigorous and active; his repetitive and apparently purposeless hand movements were jerky and irregular, and he was obviously pale but not anaemic. There was no evidence of excessive sweating, and although polyuria was suspected by the nursing staff, this was not confirmed by measurement. Nystagmoid movements of the eyes and fundal changes suggesting optic atrophy were noted during the latter months of his illness. This infant died at the age of 10 months, and at the post-mortem examination an astrocytoma of the hypothalamus was found to be invading and filling the third ventricle.

Two other infants admitted to the wards of the Royal Alexandra Hospital for Children during the past five years have presented clinical features similar to those exhibited by A. and B., and both of these infants were also found to be suffering from third ventricle neoplasms.

C. (Table I) made satisfactory progress until the age of about three months, when failure to thrive and irregular vomiting became apparent. At the age of one year she was obviously pale, but not anaemic, grossly emaciated, but surprisingly alert, happy and full of vigour. Polyuria was suspected, but not established by measurements of her urinary output, and a strange restlessness associated with excessive motor activity became particularly marked during the latter months of her illness. Signs suggesting optic atrophy were detected shortly before her death, which occurred at 16 months, and at the post-mortem examination the third ventricle was found to be partially filled by an astrocytoma.

TABLE I.

Tabulation of Clinical Features Exhibited by Four Infants Suffering from Neoplasms in the Third Ventricle Region.

Observation.	Patient.			
	A., Male.	C., Female.	D., Female.	B., Male.
Age at onset of signs .. .. .	2 months	3 months	5 months	3 months
Age at death .. .. .	19 months	16 months	17 months	10 months
Emaciation .. .. .	+++	+++	++	+++
Hyperkinesia .. .. .	+++	+	+	+
Pallor .. .. .	+	++	++	+
Vomiting .. .. .	+	+	Occasional	+
Exceptional "alertness" ..	+	+	—	+
Physical vigour .. .. .	++	++	—	++
Sweating .. .. .	++	—	+	—
Polyuria .. .. .	? +	? +	—	? +
Coarse tremor .. .. .	+	—	—	+
Nystagmoid movements ..	+	—	++	+
Optic fundi .. .. .	"Normal" from 4 to 11 months.	? Atrophic at 16 months.	? Atrophic at 15 months.	? Atrophic at 7 months.
Final diagnosis .. .. .	(I) Oligodendroglioma of optic chiasma invading the third ventricle. (II) Pyloric stenosis.	Astrocytoma of the third ventricle.	Bipolar spongioblastoma of the third ventricle.	Astrocytoma of the third ventricle.

D. (Table I and Figure II) was apparently well until the age of about five months, when occasional vomiting, progressive and unexplained loss of weight, a marked degree of pallor and excessive sweating of trunk and limbs became evident. Later this infant became very excitable and overactive, and developed nystagmoid movements of her eyes and fundal changes suggesting optic atrophy. Her death occurred at 17 months, and at the post-mortem examination a tumour (bi-polar spongioblastoma) filled most of the third ventricle.



FIGURE II.

D., aged 14 months. Final diagnosis, bipolar spongioblastoma invading the third ventricle.

A few months after B. (Case II) was admitted to the Royal Alexandra Hospital for Children, the writer was asked to see in consultation a male infant, aged 18 months, C., whose history, behaviour and physical signs strongly suggested that he might be suffering from a neoplasm in the neighbourhood of the diencephalon. This severely wasted infant's vomiting, which had begun at the age of about eight months, was associated with excessive motor activity and a surprising degree of mental alertness. At the age of 18 months, despite 10 months of irregular vomiting associated with gross and progressive emaciation, this infant was surprisingly alert and vigorous; there was no record of polyuria, but marked pallor and excessive sweating were prominent features of his illness. His ocular fundi remained normal in appearance, and no nystagmus or nystagmoid movements were noted. Ultimately the available evidence from various investigations suggested the presence of a subtentorial neoplasm associated with marked dilatation of the third ventricle, and when the child was aged a little more than two years, a mid-line cerebellar astrocytoma was removed by Mr. M. Sofer Schreiber. Rapid and dramatic recovery followed this operation, and within about five weeks the child had lost his pallor, his excessive sweating was no longer evident, his vomiting had ceased and he had gained more than 10 pounds in weight. This case may well represent another example of the "diencephalic syndrome" which is the subject of this article, but has not been included in the present series of four infants, as the neoplasm was not situated in the region of the third ventricle.

#### Discussion.

From the slender and poorly documented case records of these four infants it may be possible to suggest the following clinical syndrome.

An apparently well infant, who has made satisfactory progress for the first few months of life, begins to suffer from some irregular vomiting, fails to thrive, and very rapidly becomes grossly emaciated and obviously pallid, but not anemic. The infant usually remains exceptionally alert and surprisingly vigorous, and a variable degree of hyperkinesia is apparent from a relatively early stage of the illness. Excessive sweating may be noted, and nystagmoid movements of the eyes and signs suggesting optic atrophy may be evident during the latter months of the infant's life. Death usually occurs about 12 to 18 months after the onset of the illness, as a result of a tumour in the region of the third ventricle.

I am acutely aware of the hazards which surround any attempt to define a clinical syndrome on the basis of four incomplete and poorly documented case records; but I believe that these four case histories suggest a recognizable syndrome,

which resembles that described by Russell (1951), and which may be more clearly defined at a later date.

#### Summary.

An attempt has been made to define a clinical syndrome of early infancy caused by a neoplastic lesion in the region of the third ventricle. The outstanding features of this syndrome include irregular vomiting, which usually begins during the first trimester of life, gross and progressive emaciation, obvious pallor, exceptional alertness and physical vigour and a variable degree of hyperkinesia. Excessive sweating may be noted, and nystagmoid movements of the eyes and signs suggesting optic atrophy may be evident during the latter months of the infant's life.

#### Acknowledgements.

I wish to thank Dr. Kathleen Winning and Mr. Marcel Sofer Schreiber for permission to report the histories of three affected infants, who were admitted to the Royal Alexandra Hospital for Children under their care, and I am particularly grateful to Dr. A. Russell, of London, for his helpful letter about this problem.

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#### Reviews.

**The Family in Psychotherapy.** By C. F. Midelfort, M.D.; 1957. New York, Toronto, London: The Blakiston Division, McGraw-Hill Book Company, Incorporated. 8" x 5½", pp. 216. Price: \$6.50.

BETWEEN the extremes of individual therapy which culminated in psychoanalysis and attention to the environment with the assistance of social workers and others, some psychiatrists are treating patient *plus* family.

In "The Family in Psychotherapy", Dr. C. F. Midelfort, of the Gundersen Medical Foundation, Wisconsin, describes his methods, which take into account the ethnic, religious and cultural backgrounds of the families of his patients. His working principle is that his patients suffer from love-privation, a need which has to be met not only by giving them care, attention and personal interest, but at times also by physical contact. The psychotherapist acts as a substitute until one or more of the family have been helped by psychotherapy and counselling to give the patient the love that he lacks. Finally, after receiving love, the patient should become able to give it as well. Dr. Midelfort's methods are unusual. He has given psychotherapy to a woman in bed with her husband, and fed another with milk "as from her mother". His treatment of schizophrenia, depression, paranoid illness, psychopathic personality and psychoneurosis is illustrated by extracts from recorded interviews. In one case of a strong maternal attachment, the deferral of a military "call-up" appears to have been the main therapeutic agent. The author overlooks the fact that loss of capacity to feel and give love is symptomatic in many depressions, and normal relationship may be established after electroconvulsive therapy, independently of family attitudes and psychotherapy. When he states that "paranoid feelings of rejection are often true" and "it is not necessary for paranoid reactions to be delusional", he is surely misusing words. We do not doubt that Dr. Midelfort has a successful way with him; but it is a way to which verbal description does less than justice. Nevertheless, his emphasis on the family situation offers a lesson worth learning by psychotherapists.

**Psychology, Religion, and Human Need: A Guide for Ministers, Doctors, Teachers and Social Workers.** By W. L. Carrington, M.D.; 1957. London: The Epworth Press. 8½" x 5½", pp. 320. Price: 30s.

As is stated in the introduction, "this book is an attempt to give an account of the kind of pastoral ministry to which it is believed the Christian Church is being called in these days". The pastoral ministry is envisaged as being directed towards people's needs, and consists of pastoral visiting and interviewing, leadership in worship, teaching about marriage, parenthood and the spiritual life, personal counselling for marital, emotional and spiritual problems, and the Church's ministry to the sick. The leadership of this pastoral ministry falls mainly to ministers, and it is to them that the book

seems primarily directed. But the author rightly insists that suitably trained laymen, such as doctors, teachers and social workers, can and should help in some aspects of pastoral work, and so this book will be useful to them also as an aid to understanding both the minister's role and their own. Dr. Carrington is well known to many Australians as a writer, lecturer and broadcaster on religion and psychology. He is a Melbourne general practitioner with special interest and experience in psychiatry. He is also lecturer in pastoral psychology at Ridley College, the Anglican Theological College of Melbourne, and Chairman of the National Marriage Guidance Council of Australia. He brings to the book much wisdom gleaned from all these fields, and the result is an important contribution to the task of integrating the work of ministers, doctors, teachers, social workers and others in meeting human need.

The book is divided into four sections. The first covers briefly the scope of pastoral work, and the main principles of psychological and spiritual growth. There is a useful survey of the various schools of psychological theory, although readers who are new to this field may find it hard to follow. In the chapter on innate factors in personality growth, he mentions instinctive urges, temperament and physique, but omits the factor of intelligence. The same omission is evident in the otherwise excellent chapter on counselling, in which the client's intellectual capacity and its influence on counselling technique are not given due weight.

The second section, which deals with interviewing technique, worship, prayer, marriage and parenthood, is admirable, and the author's emphasis on equipping parents to give religious and sex education to children and adolescents in the home is most timely.

The third section discusses the healing ministry in its medical, pastoral and priestly aspects. Throughout, the author regards spiritual factors quite naturally as part of the total approach, and seeks to integrate religious ministrations with those of scientific medicine. To this end he stresses the great need of ministers for more adequate training in understanding personality in health and illness, and ways in which they can cooperate with doctors in helping the sick. He might have added that doctors need educating with regard to the role of the minister and of spiritual factors.

It should be noted that Dr. Carrington does not advocate that ministers take responsibility for treating definite neurotic disorders. These and all other mental illnesses are the responsibility of the doctor. With this in mind, he provides a chapter on the main types of mental illness to help the minister pick out these cases for referral to the doctor. However, he does indicate that doctors and ministers can work together in caring for the mentally ill, although practical ways of doing this are not discussed in detail. Incidentally, a notable omission in this chapter is epilepsy.

The final section deals with the evangelistic ministry. Ministers whom the author has warned in the previous section not to trespass on medical ground may feel that he encroaches on their field here and may quarrel with his benevolent theology. They may also be surprised that neither in this section nor in the chapter on adolescence is there any mention of the phenomena of religious conversion. Nevertheless, this last section is an inspiring record of a Christian doctor's convictions, and should help many to a clearer perception of the relevance of Christianity to all aspects of life, including health and sickness.

Most of those who read this book will be busy people, and they will therefore be grateful for its clear, orderly, compressed, readable, though didactic, style. A wealth of helpful material is presented in readily available form, and should provide a most useful basis for further discussion and work in this field.

## Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Uses of Epidemiology", by J. N. Morris, M.A., F.R.C.P., D.P.H.; 1957. Edinburgh and London: E. and S. Livingstone, Limited. 8½" x 5½", pp. 144, with illustrations. Price: 17s. 6d.

A book addressed to students of both clinical and preventive medicine, in which the author has tried "to indicate how epidemiology can serve as a means of bringing these together".

"Medicine and the Navy, 1200-1900", by J. J. Keovil, with an introduction by Sir Henry Dale; Volume I—1200-1649; 1957. Edinburgh and London: E. and S. Livingstone, Limited. 8½" x 6½", pp. 270, with several illustrations. Price: 40s.

The first volume of the first comprehensive history of naval medicine in Britain.

"A Practical Handbook of Midwifery and Gynaecology: For Students and Practitioners", by W. F. T. Haultain, O.B.E., M.C., B.A., M.B., B.Ch., F.R.C.P.Ed., F.R.C.S.Ed., F.R.C.O.G., and Clifford Kennedy, M.B., Ch.B., F.R.C.S.Ed., F.R.C.O.G., including a section on the "Management of the Infant and Neonatal Conditions", by J. L. Henderson, M.D., F.R.C.P.Ed.; Fifth Edition; 1957. Edinburgh and London: E. and S. Livingstone, Limited. 8½" x 5½", pp. 420, with 48 illustrations. Price: 30s.

The previous edition was published five years ago. The book has since been revised.

"The Year Book of Endocrinology (1956-1957 Year Book Series)", edited by Gilbert S. Gordan, M.D., Ph.D., F.A.C.P.; 1957. Chicago: The Year Book Publishers. 7½" x 5", pp. 334, with 73 illustrations. Price: \$6.75.

One of the Practical Medicine Series of Year Books.

"An Introduction to Electrocardiography", by L. Schamroth, M.B., B.Ch. (Rand.), M.R.C.P.E., F.R.F.P.S.; 1957. Oxford: Blackwell Scientific Publications. 9" x 6", pp. 72, with 106 illustrations. Price: 12s. 6d.

Written for the student, hospital medical officer and general practitioner.

"Bronchopulmonary Diseases: Basic Aspects, Diagnosis and Treatment", by 142 authors, edited by Emil A. Naclerio, M.D., with a foreword by Richard H. Overholt, M.D.; 1957. New York: Paul B. Hoeber. 10½" x 8", pp. 992, with 719 illustrations. Price: \$24.00.

A comprehensive treatment of its subject.

"Physiology of Prematurity: Transactions of the First Conference, March 21, 22 and 23, 1956, Princeton, N.J.", edited by Jonathan T. Lanman, M.D.; 1957. New York: The Josiah Macy, Jr. Foundation. 9" x 6", pp. 152, with 42 illustrations. Price: \$3.25.

Contains papers and discussions on foetal-maternal endocrinology in late pregnancy and on the foetal and placental circulation in late pregnancy.

"The Year Book of Neurology, Psychiatry and Neurosurgery (1956-1957 Year Book Series)", "Neurology", edited by Roland P. Mackay, M.D., "Psychiatry", edited by S. Bernard Wortis, M.D., "Neurosurgery", edited by Oscar Sugar, M.D.; 1957. Chicago: The Year Book Publishers. 7½" x 5", pp. 596, with 86 illustrations. Price: \$7.00.

One of the Practical Medicine Series of Year Books.

"The Medical Clinics of North America, July, 1957: Nationwide Number: The Experience of Medicine", edited by Russell L. Cecil, M.D.; 1957. Philadelphia and London: W. B. Saunders Company. Melbourne: W. Ramsay (Surgical), Limited. 8½" x 5½", pp. 261, with 109 illustrations. Price: £3 2s. 6d. per annum (cloth binding) and £6 15s. per annum (paper binding).

Contains the personal medical experiences of 20 senior medical practitioners.

"An Introduction to Electromyography", by Frits Buchtal, M.D.; 1957. Copenhagen: Gyldendal Presseafdeling, Scandinavian University Books. 9½" x 6½", pp. 44, with eight illustrations. No price stated.

Not a comprehensive treatise, but a guide for those who wish to study the subject in more detail.

"The Stress of Life", by Hans Selye, with an introduction by Sir Heneage Ogilvie; 1957. London, New York, Toronto and Melbourne: Longmans, Green and Company. 8½" x 5½", pp. 346. Price: 22s. 6d. (Australian).

Written for the general public.

"Spot Diagnosis", compiled by the editors of the *British Journal of Clinical Practice*; Volume III; 1957. London: Harvey and Blythe, Limited. 8½" x 5½", pp. 144, with many illustrations. Price: 10s. 6d. (English).

Clinical case histories and photographs presented as exercises in diagnosis.

# The Medical Journal of Australia

SATURDAY, NOVEMBER 9, 1957.

## CARDIO-RESPIRATORY FUNCTION IN MITRAL STENOSIS.

New techniques and refinements of old ones have led to remarkable advances in cardiology in recent years, and the studies of clinicians and research workers have been vastly stimulated by the intrusion of the surgeon's restless hands. In the early excitement the fact that the lungs and heart are functionally inseparable was lost sight of, and indeed one might have been forgiven for gaining the impression that the lungs were little more than an unfortunate anatomical obstacle to the passage of a catheter round most of the circulatory system. Eventually, however, the catheter became wedged in the pulmonary capillaries—with somewhat equivocal results—and latterly some attention has been paid to pulmonary function in cardiac disease. This field, studied in isolation, will not remain fertile for long, and for this reason one welcomes the appearance of the term "cardio-respiratory" employed by K. W. Donald and his associates<sup>1</sup> to describe their recent work on the pathological physiology of mitral stenosis. Perhaps the splendid isolation which the heart has enjoyed has received a rude shock from the contention of J. B. Chauvois<sup>2</sup> that even William Harvey thought that the starting-point of the circulation lay elsewhere.

Mitral stenosis has been the subject of most study, and appropriately so; it is a disabling and distressing condition which is common enough to permit the ready collection of an adequate series, and it holds out some hope that splitting the valve may reverse some of the functional abnormalities and so perhaps throw light on the mechanisms involved in their production in the first place. Of the simple tests reflecting cardio-respiratory performance, the assessment of the maximum breathing capacity in relation to the ventilatory response to exercise has perhaps the most interest and significance. J. P. P. Stock and M. C. S. Kennedy<sup>3</sup> have shown that there is a decrease in maximum breathing capacity with increasing disability, just as there is in most forms of pulmonary disease. The relationship is closer in the latter, because in these conditions the ventilatory "cost" of a standardized exercise test (they used the step test devised by Hugh-Jones<sup>4</sup>) remains more or less constant, irrespective of the severity of the disorder, and is not much above the ventilatory cost of similar exercise in normal subjects. In mitral stenosis, on the other hand, the ventilatory cost

of this exercise is considerably increased, and it increases still more with increasing severity of disability. Stock and Kennedy have found therefore that disability in mitral stenosis correlates best with an index based upon both the maximum breathing capacity and the ventilatory requirement for exercise. Put in another way, a patient with emphysema and a patient with mitral stenosis may have a similar maximum breathing capacity; the latter may prove the more disabled, because in order to perform a certain amount of work he has to approach more nearly his maximum ability to ventilate his lungs. Stock and Kennedy noted that the ventilatory cost of exercise was reduced by successful valvotomy, the maximum breathing capacity remaining unchanged. This leads to the question of why ventilatory requirement for exercise should be increased. Stock and Kennedy did not study this aspect, but they argued that as the same hyperventilation on exercise was observed in marked degree in a patient with pulmonary stenosis, radiologically oligæmic lung fields and normal arterial oxygen saturation, the hyperventilation could scarcely be attributed to nervous reflexes originating in congested lungs. J. E. Cotes<sup>5</sup> after reviewing the literature and adding experimental observations of his own, also considered that there was no need to invoke mechanisms other than those responsible for the exercise hyperpnea of normal subjects (oxygen lack, carbon dioxide production, lactic acid formation and body temperature rise). In patients with mitral stenosis there is evidence for an increased response to anoxia and to carbon dioxide. Cotes noted in particular that breathing 100% oxygen reduced the exercise ventilation of these patients much more than it did in normal subjects. He also found a correlation between rise in blood lactic acid content and mean minute volume during exercise in normal subjects; this correlation was lost in patients with mitral stenosis, but it reappeared if, in addition to the exercise ventilation, the ventilation during the recovery phase was taken into account. In patients with mitral stenosis the threshold level of exercise beyond which blood lactic acid levels tend to rise is lowered compared with that in normal individuals, probably because of the low and relatively fixed cardiac output. The abnormally high levels attained during exercise may well be a factor in the fatigue of which these patients complain, and which is in fact as important a reason for their stopping some exercise as is the development of unbearable dyspnoea.

From the clinical viewpoint the increased ventilatory requirement on exercise may be regarded as an "explanation" of exertional dyspnoea in this condition, although it is not, of course, the basic cause of the sensation. Since the work of R. V. Christie and J. C. Meakins<sup>6</sup> over twenty years ago it has been appreciated that the work of breathing is related to this sensation; Christie and his colleagues<sup>7</sup> have shown that the work of respiration is increased in patients with mitral stenosis largely owing to the increased stiffness of the lungs (low compliance). Subsequently they have suggested that in these patients, as well as in normal subjects and patients with emphysema, the feeling of dyspnoea is related particularly

<sup>1</sup> *Clin. Sc.*, 1957, 16: 325.

<sup>2</sup> "William Harvey: His Life and Times: His Discoveries: His Methods", Hutchinson, London, 1957.

<sup>3</sup> *Lancet*, 1953, 2: 5.

<sup>4</sup> *Brit. M. J.*, 1952, 1: 66.

<sup>5</sup> *Clin. Sc.*, 1955, 14: 317.

<sup>6</sup> *J. Clin. Investigation*, 1934, 13: 323.

<sup>7</sup> *Clin. Sc.*, 1954, 13: 137.

<sup>8</sup> *Ibid.*, 1954, 13: 625.

to the magnitude of the intrapleural pressure changes during the respiratory cycle. Recent studies of pulmonary compliance have been made by W. W. Pryor<sup>1</sup> and his associates and by N. R. Frank<sup>2</sup> and others. Both groups confirmed the finding that compliance is frequently low. The former group also found considerable lowering of compliance on exercise in those whose compliance was normal at rest, but no change in those whose compliance was already low. This was interpreted as indicating that pulmonary congestion lowers compliance, an observation previously made by others, but that other factors, probably including a complicating diffuse fibrosis, are also operative. Similar findings were reported by Frank and his co-workers, who also showed a significant correlation between compliance and vital capacity or total lung capacity. Their paper summarizes current views on some of the factors affecting compliance, stressing in particular that a reduction in lung volume, irrespective of cause, will have the effect of lowering compliance.

A valuable paper has recently been published by K. W. Donald, J. M. Bishop, O. L. Wade and P. N. Wormald<sup>3</sup> dealing with cardiorespiratory function in 28 patients before and two years after mitral valvotomy. They present a tantalizingly large amount of information, from a battery of investigations, which lends itself poorly to summary, especially as it is difficult to formulate any unifying hypothesis to cover all the findings. Of immediate practical importance is their conclusion that, in spite of marked clinical improvement after operation, the cardiac output and pulmonary vascular pressures remained abnormal, especially on exercise. On the other hand, considerable improvement was indicated by the fall in both resting and exercise ventilation, by the decrease in pulmonary vascular resistance and, perhaps most important of all, by the diminution to virtually normal levels at rest in right ventricular work (a function of the reduced cardiac output as much as the fall in vascular resistance). The decrease in cardiac output paralleled the fall in oxygen uptake observed in the post-operative studies, an observation which was taken to mean that the high pre-operative oxygen uptake reflected the oxygen cost of the increased work of ventilation in these patients. This correlation is not apparent in the results reported by J. A. Wood and others, but these workers did find a similar decrease in exercise ventilation. Donald and his colleagues found a relationship between resting ventilation and pulmonary capillary (wedge) pressure which is of interest in relation to the compliance studies previously mentioned. The suggestion is made that the excessive exercise ventilation observed pre-operatively may be related to small falls in arterial oxygen partial pressure during exercise (it is likely, incidentally, that in some cases of mitral stenosis there is some reduction in the diffusing capacity for oxygen); these falls are much smaller on exercise after valvotomy. Donald's group also suggests that there may be an increased response to carbon dioxide production, as occurs when hyperpnoea at rest has previously lowered the blood content of carbon dioxide and base. They point out that, as cardiac output on exercise after valvotomy shows no significant improvement in some patients whose

exercise ventilation nevertheless falls greatly, blood lactic acid levels are unlikely to be the chief factor in the increased ventilatory requirements of exercise.

In summary, symptomatically, the patient's improvement is reflected in a fall in exercise ventilation; physiologically, it is perhaps best expressed in terms of the diminution in right ventricular work at rest. The fact unfortunately remains that the haemodynamics of the lesser circulation in particular stays distinctly abnormal after apparently highly successful surgical treatment. For this reason, and because of our incomplete understanding of the factors involved, Donald and his associates do not believe that the waning enthusiasm for cardiac catheterization in the routine assessment of patients with mitral stenosis is wholly justified. Donald is well qualified to express this opinion, possibly an unpopular one at the present time, for few have made such comprehensive observations and few have used them not merely as objective confirmatory evidence of altered clinical status but also as a means to a physiological end. At all events it is sincerely to be hoped that his cooperative volunteers will submit themselves once more to his investigations in a few years' time.

## Current Comment.

### THE EFFECT OF A CIGARETTE ON AIRWAYS RESISTANCE IN EMPHYSEMA.

Those addicted to tobacco in one or other of its forms have been threatened in the past with death, torture, banishment and excommunication. Severe penalties have been prescribed by Christian and Mohammedan authorities and by Russian, Turkish, Persian, Indian, French and Chinese rulers; King James I compromised with his famous "Counterblaste": "Have you not reason, then, to be ashamed and to forbear this filthy novelty? . . . In the abuse thereof sinning against God, harming yourselves both in persons and goods . . . a custom dangerous to the lungs, and in the black stinking fume thereof, nearest resembling the horrible Stygian pit that is bottomless." Neither deeds nor words hindered significantly the growth of the weed, and time had its usual effect of producing tolerance, or somnolence, in society. The recrudescence of a centuries' old controversy is unlikely in itself to win many converts to the cause of the "anti-tobacconists", but, with any luck, time and the perpetual motion of the pendulum of fashion may win the battle for them, at least for a century or so. The statistical threat of cancer, tuberculosis and chronic bronchitis seems to be no more effective as a deterrent than having one's nose slit, one's head chopped off or being sent to Siberia. This may appear a rather tortuous introduction to a much more mundane aspect of the evils of smoking, but it may well be that practical advice based upon the latter may prove a more telling argument with some patients than comparatively vague hints of dire events to come.

Using an experimental design admirably planned to avoid errors of various types, R. H. Eich and his colleagues<sup>4</sup> have studied the effect of a single cigarette upon pulmonary mechanics in habitual smokers with and without emphysema. They found no significant change in compliance, as might be expected, but they found an unequivocal increase in non-elastic resistance, which was almost certainly due to an increase in airways resistance. This change was associated with an increase in functional residual capacity, which is a recognized sequel to an overall reduction in bronchial calibre. It may be noted that these effects were demonstrated in 14 out of 15 emphysematous subjects and were not apparent in a con-

<sup>1</sup> *Circulation*, 1957, 15: 721.

<sup>2</sup> *Am. J. Med.*, 1957, 22: 516.

<sup>3</sup> *Circulation*, 1956, 13: 178.

<sup>4</sup> *Am. Rev. Tuberc.*, 1957, 76: 22 (July).

trol series. Two patients were conscious of increased dyspnoea after smoking; as the authors point out, increase in airways resistance is closely related to disability.

Eich and his associates have provided useful material for the physician's counterblast, at least in the case of patients with emphysema, and perhaps, by analogy, bronchitis and asthma. Whether the assurance that smoking makes the patient's breathing worse—a fact which he is sometimes honest enough to admit—will be any more effective than iatrogenic cancerphobia or moral exhortation remains to be seen. Those who prefer the dogmatic assurance approach are grateful to Eich's work for providing objective evidence for their contention.

#### TANTALUM MESH IN THE REPAIR OF LARGE VENTRAL HERNIAS.

W. REMINE AND R. WHITE<sup>1</sup> are convinced that tantalum gauze offers a satisfactory and convenient method of repairing large hernial defects. They point out that it is less cumbersome than cutis grafts and fascial transplants and offers no discomfort to the patient. In support of this statement they refer to the experimental and clinical work of A. R. Koonitz,<sup>2</sup> of Koonitz and R. C. Kimberley,<sup>3</sup> and of T. Throckmorton.<sup>4</sup> These authors consider that it does not matter if the gauze fractures by "work hardening", as the strength of the repair lies in the fibroblastic response around and through the tantalum mesh and not in the mesh itself.

ReMine and White speak with some authority, as they refer to a review of 2000 ventral hernial repairs at the Mayo Clinic from 1945 to 1953, of which 31 were effected with tantalum mesh. The average age of the patients in these 31 cases was 58 years. Twenty-five were overweight, the mean average being 25 pounds, which points the way to one obvious pre-operative measure—namely, the reduction of overweight, preferably by dietetic measures. It is better thus to try to diminish the amount of some of the intraabdominal contents rather than by the induction of a prolonged pneumoperitoneum to stretch the abdominal wall or the even more radical method of bowel resection advocated by some authorities.

Of the patients concerned 17 had had previous repairs, one having had five previous attempts at repair, whereas after the tantalum mesh repair there was no recurrence in this patient three years later. Nine patients were seen in emergency with acute intestinal obstruction, and of these two died as a result of complications from the intestinal obstruction. These were the only two deaths in this series of tantalum mesh repairs. As a consequence it is obviously unwise to utilize the tantalum at the same time as one operates for intestinal obstruction. The repair can always be done later.

At a follow-up investigation, information was obtained on the survivors. Two died of causes unrelated to the hernia, and they had had no recurrence. The tantalum gauze had to be removed in two cases, once for recurrence and once for a persistent draining sinus thought to be due to the buried silk sutures used to keep the mesh in place. Seven of the 27 hernias recurred, all within nine months. Of 27 patients, 20 were well without recurrence from one to nine years after surgical repair.

The causes of the recurrences are not stated in this review, but it is known, as with other hernias, that if these patients go back to heavy work, such as industrial or waterside work involving heavy lifting, then a high percentage of recurrence will follow. Tantalum and its fibrous tissue reaction cannot and do not stop recurrence in such circumstances.

Thus from this series from the Mayo Clinic and from the experience of others the opinion is formed that, whilst tantalum may be the best material so far available in the

repair of large ventral hernias, its use is not yet the final answer; tantalum still has certain deficiencies quite apart from haematoma formation in the wound and staphylococcal infection of the wound, the latter often though not necessarily being dependent on the presence of the former. From the usual extensive dissection required one might expect these complications more frequently than they actually occur. However, even if infection does occur, removal of the gauze is rarely required, provided one has used tantalum or nylon sutures to keep the gauze in place and not silk. Haematomas must be evacuated quickly, as one round the mesh may prevent the fibrous tissue from growing through the mesh. In this regard, the dressing of the wound with the volatile plastic "Nobecutane" allows frequent post-operative inspection of the wound without adding the risk of cross-infection.

The deficiencies of tantalum mesh repair are several. The first is that recurrence does take place, as the Mayo Clinic and other series show. This recurrence results from the hernia bulging around the edge of the tantalum; so that eventually one has the hernia again, and on its summit is the tantalum with its fibrous tissue plaque, still pliable but not elastic. Operation for removal of the gauze may be extremely difficult because of this fibrous tissue reaction. The second deficiency is that serous fluid reaction may occur around the tantalum and drain through the wound for weeks or even months, and at later operation one may find a large cystic space around the tantalum lined by thin endothelium, with no fibrous tissue reaction going through the mesh. A similar reaction can occur, of course, in any area where extensive tissue planes have been opened, especially after the repair of ventral hernias without tantalum gauze or any other prosthesis. This reaction, however, requires removal of the tantalum. The third drawback is that necrosis of the skin superficial to the gauze can occur, and the skin quietly disappears; whilst sometimes granulation tissue grows up and fills in the hiatus with scar tissue, at times this does not occur, and the tantalum must then be removed. Finally, the underlying peritoneum may become necrotic and disappear, and this may be disastrous. Although ReMine and White consider that tantalum mesh when in direct contact with bowel forms only slight adhesions and consider this to be a desirable attribute, another thing can happen, and that is that the edge of the tantalum may actually ulcerate into the bowel lumen, and when the tantalum is removed the bowel is perforated. Thus it is obvious that to put tantalum mesh in direct contact with bowel without a layer of peritoneum or fascia intervening is most undesirable.

Consequently, it is clear that tantalum has a place in the repair of certain large hernias which cannot be repaired by fascial flaps from local sources, but one must expect a certain number of recurrences after repair by tantalum or similar plastic prostheses, all of which have their own undesirable side effects.

#### COMPLICATIONS DUE TO THE JOHNSON URETERAL STONE BASKET.

A RECENT REPORT by J. W. Barloon<sup>1</sup> provides details of two cases of impaction of the Johnson ureteral "stone basket" in the lower part of the ureter, necessitating open surgical removal. He states that, although this instrument is a widely accepted and useful one for extracting small calculi from the lower third of the ureter, its use may not be without penalty in some cases. In both the cases reported, in spite of careful open surgical approach to remove the impacted "basket", a stricture has developed at the site of damage. However, instead of condemning this barbarous and unnecessary instrument, the author concludes that it is most useful for stones less than five millimetres in diameter, and, of course, lying in the lower third of the ureter. It is some consolation that he gives a warning that in other areas of the duct or with longer stones, it may be dangerous.

<sup>1</sup> *Ann. J. Surg.*, 1957, 93:1008 (June).

<sup>2</sup> *J. Internat. Coll. Surgeons*, 1951, 16:637 (November).

<sup>3</sup> *Ann. Surg.*, 1950, 131:666 (May).

<sup>4</sup> *Surgery*, 1948, 23:32 (January).

<sup>1</sup> *J. Urol.*, 1957, 77:151 (February).

## Abstracts from Medical Literature.

### OPHTHALMOLOGY.

#### Simple Operation for Senile Spastic Entropion.

M. BODIAN (*Am. J. Ophthalm.*, July, 1957) describes a simple technique for the cure of spastic entropion. A skin incision parallel to the lid margin is made two or three millimetres below the margin and for its entire length. The lower skin margin is undermined to expose the orbicularis. Two parallel rows of diathermy coagulation are then made two and five millimetres below the line of incision. A strip of skin three or four millimetres in width is then excised from the lower wound edge, and the skin is closed with interrupted plain 4-0 catgut sutures.

#### Deep Forms of Herpetic Keratitis.

P. THYGESON AND S. J. KIMURA (*Am. J. Ophthalm.*, April, 1957) describe the deep forms of keratitis which may occur in a herpes simplex infection. The classical example of deep involvement in herpetic keratitis is disciform keratitis. In a benign case, the lesion is characterized by intense oedema of the stromal fibres, limited to the central two-thirds of the cornea and unaccompanied by any major necrosis of the stromal fibres or even by any new vessel formation. In such cases the condition tends to run a course of two to three months, and heals with minimal scarring and little or no diminution of vision. A more common type results in dense scar formation and sometimes corneal necrosis with perforation. This may last for many months and reduces vision. A troublesome complication is secondary glaucoma. An extension of the disciform lesion may produce interstitial keratitis. Hypopyon keratitis may occur especially in those treated with the steroids. Diagnosis is easy if the patient has been followed from the onset of the dendritic lesion. In late cases the presence of corneal anaesthesia and retention of the ulcer in part of the characteristic amoeboid configuration give the diagnosis. Herpetic iridocyclitis is common in deep stromal keratitis, and in fact such iridocyclitis may occur in the absence of keratitis. The authors have shown that topical steroid therapy often increases the chronicity of the disease, the necrosis of the stromal fibres and the incidence of uveal complications, and such therapy is definitely contraindicated in herpes simplex infection.

#### Superficial Forms of Herpetic Keratitis.

H. L. ORMSBY (*Am. J. Ophthalm.*, April, 1957) discusses the superficial forms of keratitis which may occur in herpes simplex. In the early stages a vesicle appears and enlarges in a series of branching processes to form the typical dendrite. Variations in the corneal picture are due to the strain and virulence of the infecting agent, the degree of local immunity of the cornea, the presence of circulating antibodies, the duration of the disease, the history of previous attacks, the effect of treatment and the presence or absence of secondary infection.

Acute follicular conjunctivitis is an invariable accompaniment in children. In adults with circulating humoral antibodies to the virus, a follicular reaction seldom accompanies dendritic ulceration. Cauterization of the dendritic ulcer with iodine together with denudation of the epithelium is effective in the treatment of superficial lesions.

#### Acute Herpetic Keratoconjunctivitis.

A. E. BRALEY (*Am. J. Ophthalm.*, April, 1957) describes acute herpetic keratoconjunctivitis as a disease of young children and teenagers. It usually begins as unilateral acute conjunctivitis. At times there are small vesicles on the cilia line which develop into small ulcers. There may be local dermatitis of the eyelid. Herpes labialis and stomatitis are sometimes present, especially in infants and young children. The conjunctivitis may be part of a generalized herpetic infection which may involve the face. The keratitis, at first diffuse, develops a dendritic pattern as the disease progresses, and corneal sensitivity is lost. This is an important diagnostic point in early phases of the disease before a definite dendritic pattern is developed.

#### The Present Status of the Viral Keratoconjunctivitis Problem.

P. THYGESON (*Am. J. Ophthalm.*, April, 1957) comments on the various types of conjunctivitis and keratitis known to be or suspected of being due to viruses. The causative agent in trachoma has been classified with the psittacosis-lymphogranuloma group. The sulphonamides and broad-spectrum antibiotics are successful in the treatment of individual cases. The virus has resisted attempts at cultivation. Inclusion blennorrhoea, which occurs in the new-born, is closely related to trachoma, but displays none of its corneal or cicatricial complications; like trachoma, it is susceptible to sulphonamides. Epidemic keratoconjunctivitis appears typically in an acute form. The corneal infiltrates may persist for months and finally disappear without scar formation. Minor epidemics are always occurring, and major epidemics can always occur. Pharyngo-conjunctival fever occurs chiefly in children, and has a relationship to swimming-pool spread. It is to be differentiated from Béal's acute follicular conjunctivitis and epidemic keratoconjunctivitis. Newcastle disease conjunctivitis, often with preauricular adenopathy, is of short duration, and is occasionally seen among poultry farmers. The disease is benign, self-limited and without corneal complication. Lymphogranuloma venereum keratoconjunctivitis is rare, and if recognized early, amenable to chemotherapy. Herpetic keratitis and keratoconjunctivitis is most serious in the cornea and uveal tract because of its chronicity and tendency to form scars. Keratitis due to variola and vaccinia is now rarely seen; the corneal lesion is disciform keratitis which often results in dense cicatrization. Keratoconjunctivitis of measles is benign and transient, and is of minimal economic and visual importance. Molluscum contagiosum on the lid produces chronic follicular conjunctivitis which, if allowed to persist, is complicated by keratitis. Extirpation of the lid margin lesion produces a cure. Herpes zoster is often associated with

keratitis, iridocyclitis and secondary glaucoma. In many severe cases, disciform keratitis develops. Other conjunctivitis and keratoconjunctivitis of suspected viral origin are Béal's acute follicular conjunctivitis, chronic follicular conjunctivitis, superficial punctate keratitis, ocular pemphigus, Reiter's disease conjunctivitis and conjunctivitis of the erythema multiforme syndrome.

#### The Treatment of Retinoblastoma by Irradiation and Triethylene Melamine.

A. B. REESE *et alii* (*Am. J. Ophthalm.*, June, 1957) briefly give their experiences in the treatment of retinoblastoma (i) by X radiation alone, (ii) by X radiation and TEM given by mouth, (iii) by X radiation and TEM given intramuscularly and intraarterially. Twenty-two eyes were treated by X radiation alone, and in these the cure was 50%. The dosage varied from 6800 to 4800r. Fifty-seven eyes were treated by X radiation and TEM given by mouth; the first dose of TEM was administered 24 hours before irradiation was begun, the second dose coincided with the last radiation treatment, and the third dose was given after irradiation was completed; the dose of X rays to these eyes was 2400r. The first 20 eyes so treated have a 70% cure rate. The oral use of TEM has been discarded in favour of the intramuscular route. The effective intramuscular dose is about one-third to one-half the oral dose. To date the authors have treated eight patients with TEM by intramuscular injection and X radiation, with arrest of the tumour. TEM was instilled into the internal carotid artery on the side of the eye involved when the tumour was very large and enucleation was refused, when tumour cells were seen histologically at the end of the optic nerve and when the orbital recurrence followed enucleation. In addition, irradiation was given and if necessary exenteration was also performed.

#### The Modern Treatment of Uveitis.

R. H. NIELSEN AND T. J. KIRBY (*Arch. Ophthalm.*, July, 1957) review the treatment of uveitis. Specific treatment is directed towards the aetiological cause—for example, syphilis, tuberculosis, brucellosis, toxoplasmosis, trypanosomiasis, actinomycosis, blastomycosis and histoplasmosis. Non-specific measures include measures to improve the general health, and local measures, which comprise mydriatics, heat, paracentesis and irradiation, and the use of non-specific drugs and chemical compounds. The last-mentioned include salicylates, antibiotics, nitrogen mustard, copper lactate given intravenously, trypsin given intramuscularly, and phenylbutazone. Finally, the use of fever therapy and the steroids is outlined.

#### "Diamox" in the Treatment of Glaucoma.

P. A. CHANDLER (*Arch. Ophthalm.*, May, 1957) gives a warning against the use of "Diamox" in angle-closure glaucoma, except in the immediate pre-operative period and as a post-operative treatment for residual glaucoma. Its greatest field of usefulness is in secondary glaucoma, and it may be helpful in open-angle glaucoma. If "Diamox" is used with

miosis in angle-closure glaucoma, the level of tension is no longer a guide to the extent of closure of the angle. "Diamox" may reduce tension, and yet the angle remain closed and permanent anterior synechiae form. The effect of "Diamox" then may be to reduce tension by reducing the formation of aqueous, and at the same time the angle is becoming closed by synechiae. Chandler compares the action of "Diamox" in angle-closure glaucoma to that of morphine in abdominal pain, in that they both mask the danger signs.

## OTO-RHINO-LARYNGOLOGY.

### Transtympanic Mobilization of the Stapes.

A. A. SCHERR (*Arch. Otolaryng.*, March, 1957) states that the results obtained in 500 cases of transtympanic mobilization of the stapes during two and a half years warrant continuation of the operation for the relief of otosclerotic deafness. Early in this class of work, a figure of 36% of first-class results was established. It has not been possible to improve on this figure. There is a recurrence of hearing loss in 20% of cases, in approximately half of which improvement follows a revision procedure. Tinnitus will stop or be greatly reduced with a successful mobilization operation.

### Factors Influencing Caloric Nystagmus in Normal Subjects.

J. MAHONEY, W. L. HARLAN AND R. G. BICKFORD (*Arch. Otolaryng.*, July, 1957) state that clinical experience has shown that the response to caloric stimulation may vary in the same person in the hands of different examiners and on different days. The measurable factors of caloric nystagmus are its latency, its amplitude, its frequency and its duration. It is difficult to determine these factors accurately by simple direct observation. An attempt has been made to obviate this difficulty by graphically recording the results. As with other reflex mechanisms, the caloric response is subject to integrative neural influences arising from the cerebrum, the cerebellum and the upper part of the cervical cord. The state of visual fixation may play an important role in the nature of the nystagmic response obtained by caloric stimulation. In addition, the influence of other cerebral factors such as concentration and diversion is to be determined. The eye is a charged body, the cornea being electropositive in relation to the retina. When electrodes are placed on either side of the eye, any movement in the plane of the electrodes will register a difference in potential across them. This small potential can be amplified and recorded. This method is advantageous, in that it permits recording when the eyes are closed. The results reveal that there is an inverse relationship between the amplitude and the frequency of nystagmus. The amplitude increases and the frequency decreases progressively with increasing interference with fixation. There is also a moderate parallel increase in the duration of nystagmus with decrease in the fixation factor. Eye closure increases these changes. Under this condition, caloric nystagmus is often

completely inhibited or is present only intermittently, appearing in brief bursts. Mental activity or alerting frequently elicits a brief burst of nystagmus when this has been inhibited by eye closure. This finding suggests that cerebral factors play an important role in caloric nystagmus, and supports the suggestion of others that the caloric response may vary with the patient's state of alertness.

### The Dry Physiologically Perfected Fenestration Technique.

J. LEMPERT (*Arch. Otolaryng.*, July, 1957) states that to make the fenestration procedure entirely successful, it has been necessary to find definite means to prevent post-operative osteogenic closure of the newly created fenestra, and to avoid post-operative inflammatory reactions in the vestibular and cochlear labyrinth, which are likely to cause impairment of hearing. The prevention of osteogenic closure has been further assured by deliberate invagination of portion of the tympano-meatal flap into the fenestra so that this may become adherent to the rim, when it consistently prevents osteogenic closure of the fenestra. Prevention of post-operative inflammatory reactions within the labyrinth called for consideration of methods to avoid the entry of blood into the perilymph space during the fenestration operation, for it had been shown in experiments on monkeys that varied degrees of post-operative labyrinthitis could be detected after permitting different amounts of blood to enter the perilymph space. Local and general analgesia rather than general anaesthesia, and strict control of all bleeding before the labyrinth was opened, assured this requirement. The author questioned whether bleeding into the perilymph space was the only cause of inflammatory reactions, since in many cases, when it was certain that no blood had entered the labyrinth, the symptoms of serous labyrinthitis nevertheless occurred in various degrees of severity. Another technical factor thought possibly to be responsible was trauma to the membranous labyrinth in attempts to remove bone chips from the fenestra after any form of pulverizing technique in making the opening. Even when this was avoided by using the cupola-lifting technique which eliminated bony fragments, some post-operative labyrinthitis nevertheless still occurred. It occurred to the author that irrigation and suction with any of the solutions used to date might be the technical error responsible for the labyrinthine reactions, since irrigation and suction must lead to replacement of some of the normal labyrinthine fluid with some of the irrigating solution. Both the mechanical irritation of the suction force and the chemical differences between the fluids used and human perilymph may be responsible for some inflammatory reactions. Tests were carried out on a series of 100 subjects, irrigation being used or avoided in alternate cases. The two proven causes of post-operative inflammation, entry of blood to the labyrinth and probing to remove bony particles, were carefully prevented. In the 50 cases in which irrigation and suction were not used after opening the perilymph space, there was no clinical evidence of post-operative labyrinthine

inflammatory reactions. In all the 50 cases in which irrigation was used, signs of reactions in various degrees of severity were found. It is concluded that irrigation and suction, after the perilymph space has been laid open, are the constant cause of post-operative inflammatory reactions. However, the need for irrigation and suction after opening the labyrinth can be avoided only by technically performing the operation without creating the need for it, by carefully removing all bone debris before making the fenestra, and by employing the cupola technique instead of a pulverizing procedure for opening the perilymph space. In summary, the following technical precautions are required: methods of anaesthesia which lessen bleeding and then control of every individual bleeding point; careful sculpture of the inner table of the mastoid recess and careful fitting of the tympano-meatal flap to it; removal of all bone dust before the perilymph space is opened; avoidance of the need to remove bone chips from the perilymph space by employment of the cupola technique instead of pulverizing the bone; complete avoidance of irrigation after the perilymph space has been opened.

### Acetazolamide in Ménière's Disease.

M. K. MUIR (*Arch. Otolaryng.*, June, 1957) states that there is a great difference between the sodium concentrations in the respective labyrinthine fluids, the level in the endolymph being always several times lower than that of the perilymph. Estimates of the sodium content of perilymph shortly after injection reveal a concentration approximately that of the blood, and suggest that this fluid does not originate from the cerebrospinal fluid, but rather comes from the blood vessels. The endolymph has a low sodium concentration. These findings favour the ultrafiltration theory. There must therefore be a barrier between the capillaries and the endolymph. This must be the epithelium of the *stria vascularis*. It may be that vasomotor labyrinthine ischaemia precedes the labyrinthine hydrops. Carbonic anhydrase, a zinc-containing enzyme, is believed to be present in every physiological barrier where ion exchange occurs. Its metabolic role is to preserve the alkalinity of sodium and potassium. It acts to retain sodium in the aqueous humour of the eye, and may have some influence in changes of intraocular pressure. Acetazolamide ("Diamox") is a carbonic anhydrase inhibitor. This was applied successfully in the treatment of ocular glaucoma. On the basis of a possibility of similar development of endolymphatic hydrops of the labyrinth, acetazolamide was used by intravenous infusion as well as by mouth in the treatment of 27 patients with Ménière's disease. It is suggested that carbonic anhydrase is in the first place responsible for sodium accumulation in the endolymph causing osmotic traction of the endolymph towards the perilymph, so that there is an increase in volume of the endolymphatic fluid, and thus hypertension. In all cases the establishment of labyrinthine reflexes was observed during treatment, and otolithic function was restored. Deafness was not much relieved, but vertigo was speedily controlled.

## Special Article.

### MEDICAL ASPECTS OF NUCLEAR RADIATION.

The following article is reprinted from the *Civil Defense Technical Bulletin*, July, 1956, a publication of the United States Federal Civil Defense Administration. It is reproduced with the permission of the United States Federal Civil Defense Administration and with the cooperation of the New South Wales Civil Defence Organization.

The initial radiation hazard from a nuclear weapon detonated high in the air is due to gamma rays and neutrons liberated at the time of the explosion, and gamma rays from the ascending cloud. Casualties from a low yield weapon will be from blast, heat, and initial nuclear radiation.

With high yield weapons the relative hazard of initial radiation is greatly reduced because the area covered by the blast and heat effects is larger than that covered by initial radiation. These weapons detonated near the surface of the earth cause great amounts of material to be drawn up into the fireball. This material is contaminated by radioactive products of the bomb. The radioactive particles will fall out, causing contamination which may be lethal over thousands of square miles. Under these circumstances radiation casualties may equal or exceed those from blast and heat.

The hazards from fallout radiation are whole-body penetrating radiation, skin contamination, and internal absorption of radioactive materials. The whole-body penetration is almost entirely gamma, since there are no neutrons present. In skin contamination, the greatest part of the dose is due to the beta component, since the beta particles are absorbed almost entirely in the layers of the skin. The internal radiation hazard results from entry of radioactive substances into the body by breathing, swallowing, or through breaks in the skin. These substances may emit beta and gamma radiation from the fission products and alpha particles from unfissioned material. Inhalation will not be significant unless the particle size is very small—0.5 to 5 microns in diameter. Ingestion and entry through wounds is not so dependent on particle size.

Radiation damage from ingested materials results from irradiation of the body—principally the gastrointestinal tract, thyroid gland, and bone—from fallout particles in the intestinal tract, and radioactive materials absorbed and remaining in the body. It is improbable that there would be enough material inhaled or swallowed during the first few days to contribute appreciably to the acute clinical problem. Chronic exposure will be discussed later.

#### The Acute Radiation Syndrome.

The most reliable means of estimating the seriousness of radiation injury is by the physician's evaluation of clinical symptoms, particularly on the day of exposure. The gastrointestinal tract is one of the most radiosensitive organ systems. Observable functional changes occur promptly after the damage has taken place. The incidence, severity, and time of beginning of vomiting and diarrhea have been shown to be a good index of the degree of radiation damage. Information on the distance from the explosion, amount of shielding present, and amount of radiation indicated by dosimeters should be taken into consideration when estimating total exposure.

On the basis of the severity and time of occurrence of gastrointestinal symptoms, casualties may be divided into three groups:

**Group I.—Survival improbable.** These will have received supralethal amounts of radiation, probably in excess of 800 r<sup>1</sup> gamma dose of whole body irradiation, in a short period. Severe, and more or less continuous, vomiting will occur within a few hours, and will be followed by diarrhea, producing severe dehydration and apathy. Death may be expected to occur at any time from one day to two weeks.

**Group II.—Survival questionable.** These will have received a dose of probably 200 r to 800 r. Vomiting will occur on the first day, but will subside within about 24 hours to be followed by a period of relative well-being from one to three weeks. This quiescent period may be followed by the development of small subcutaneous hemorrhages, sore

mouth and throat, loss of hair, bloody diarrhea, loss of weight, and infection of the thermal burns and other wounds which had been healing. Most of these symptoms are common to a variety of diseases, but the occurrence of hemorrhagic spots and falling hair are strongly suggestive of severe radiation injury.

**Group III.—Survival probable.** These will have received a dose probably less than 200 r. No symptoms are to be expected on the first day except transient nausea, some vomiting, and fatigue. Later on, from the second to fourth week, there may be a slight feeling of ill health, but no incapacity will occur in most cases.

It is obvious that it is the Group II patient who will benefit most from treatment. If thermal burns or other injuries are present, the prognosis must be looked upon with more concern.

Blood changes provide valuable information on dosage. In Group I cases there will be a profound and prompt drop in the number of white blood cells. Blood concentration may be marked due to excessive fluid loss and lowered fluid intake. However, gastrointestinal symptoms will far overshadow the blood picture.

It is Group II and III cases in which the blood picture is of great medical importance. Doses in the range of 200 r to 800 r will result in profound drop within a day or two in certain of the white blood cells. Other white blood cells are reduced in number within a week or two. The red blood cells and platelets are also depressed, and profound anemia may occur. Examination of the white blood cells soon after exposure in the low dose range is a valuable aid to diagnosis. The platelet count is correlated with the dose, and if made properly is very helpful in making the prognosis. The blood findings return to normal within one year.

The acute radiation syndrome presents no new features that are not observed daily in clinical practice. The diarrhea, dehydration, electrolyte imbalance, hemorrhage, anemia, and infection, are all commonplace findings, and the exercise of sound clinical judgement and good nursing care in the treatment of various aspects of the syndrome will save many lives and hasten convalescence.

Gastrointestinal symptoms should receive careful attention. Straining associated with diarrhea may be treated with antispasmodics such as atropine. Dehydration and electrolyte imbalance should be vigorously combated with intravenous fluids and fluids by rectum if conditions permit. A bland, soft, or liquid high-calorie diet with vitamin supplements and ample fluids should be given as tolerated. Careful attention must be given to oral hygiene.

The treatment of infections associated with radiation injury is one of the most important aspects of handling these cases. The use of antibiotics presents a problem because early prophylactic use may result in development of resistant strains of organisms when at a later time antibiotics are urgently needed. Vigorous use of antibiotics should be started immediately on development of clinical evidence of either localized or generalized infection. It is advisable to use broad spectrum antibiotics with alternating schedules.

Blood transfusions should not be given as a routine measure, but may be lifesaving when clinically indicated.

Many substances have been tested in the hope of developing a specific preventive or therapeutic agent. To date none has been sufficiently established to warrant its inclusion in the physician's armamentarium, but research continues with the hope that a specific therapy will be discovered and developed to allow a greater number of cases to recover, and the illness to be shortened.

#### Skin Lesions.

A wide variety of skin lesions may develop, depending upon variable physical as well as biological factors. The more important physical factors are:

- The earlier the contamination after the explosion, the greater the dose for a given amount of fallout material.
- The longer radioactive material remains on the skin, the greater the dose.
- The greater the proportion of high energy beta radiation, the deeper the effect.
- The distribution of material on the skin will determine the location of the lesions.

Among the biological factors are:

- Areas of the body covered by thinner skin will be more severely affected.

<sup>1</sup>Roentgen (r).—A unit of radiation quantity, defined as that amount of X- or gamma radiation which produces one electrostatic unit of charge of either sign in one cubic centimeter of air at standard temperature and pressure.

(b) Where the hair is thicker, the material will tend to remain longer, and the effect will be more pronounced.

(c) The material tends to collect and remain in areas of greater perspiration.

Preventive measures such as taking shelter or keeping indoors during the time fallout is taking place, or covering as much of the body as possible followed by removing outer clothing and washing exposed parts of the body, may completely eliminate or greatly reduce incidence and severity of skin lesions.

During the first few weeks after exposure, there may be no indications of skin damage. In more severe exposures, symptoms will occur within the first 24 to 48 hours. These will include: itching, burning or tingling sensations, and burning and watering of the eyes. Areas of redness, swelling, or blanching may be noticed. The greater the exposure, the earlier the symptoms will appear.

Within a few days early symptoms temporarily subside or disappear. The length of time before lesions develop is related roughly to the severity of exposure, and may vary from a few days following severe exposure to a few weeks after mild exposure.

Following this period of quiescence there is a recurrence or intensification of symptoms. Lesions are usually absent on areas protected by even the scantiest of clothing. They are more likely to appear on areas where the skin is thin, moist, or hairy—head, neck, armpits, and elbows. New lesions may appear over several weeks.

Reddening of the skin, spots, pimples, or raised plaques or tanning of the skin may be the first indication of damage. These lesions may coalesce and form dry, thickened, pigmented areas. Itching, burning, and mild pain may be experienced. Milder lesions may merely show dry scaling from the center outward, leaving a depigmented thinned skin, followed within a few weeks by healing and repigmentation. More severe lesions may show deep destruction with raw, weeping ulcers. Secondary infection may occur, especially if there is damage to blood cells incident to gamma radiation and if lesions do not receive proper care. These deeper lesions may be quite painful with resulting limitation of motion. The healing process may be slow or incomplete.

Loss of hair begins 2 to 3 weeks after exposure and usually involves the head. Eyebrows, eyelashes, axillary, and pubic hair appear to be more resistant. Unless the exposure has been severe, complete regrowth with normal color and texture within 5 to 6 months is to be expected.

Treatment during the acute stage is very similar to the treatment of thermal burns. Mild lesions require daily cleansing with soap and water and application of bland ointments or lotions such as calomine lotion with 1% phenol. Ulcerating lesions should be kept cleaned and dressed. Antibiotics orally, locally, or by injection should be used if secondary infection occurs, or prophylactically if there has been a high dose of gamma radiation and severe leukopenia is present. Surgical treatment may be necessary and early skin grafting may be considered in the most severe cases.

#### Late Effects.

Late effects are those harmful results which do not interfere with working efficiency during the first few weeks after exposure. They should never be thought of lightly and may be a major consideration in postattack and recovery periods. During the attack phase, however, we will be much more concerned with problems of our immediate survival and decisions on permissible radiation exposure will be made accordingly. Except for cataract development from neutron exposure and skin cancer from local radiation damage, late results from single exposures are not qualitatively different from those due to chronic exposure.

#### Chronic Radiation Injury.

Chronic injury may result from one or a combination of the following types of exposure:

- Continuous low level exposure to external sources.
- Intermittent exposure to external sources.
- Prolonged exposure to internal sources.

There is no human and very little animal experience in continuous low level exposure to radiation from external sources. There is, however, much information on long continued intermittent exposure, and little reason to doubt that the clinical picture would be indistinguishable, one from

the other. The principal findings are blood changes with leukemia predominating, skin cancer, as noted on the hands and face of radiologists, and shortening the life span—statistically demonstrable in animals and men. There will also be some genetic changes.

Prolonged exposure to internal sources may occur through continued inhalation or ingestion of radioactive substances or the fixation of long lived radiolotopes in the body. Certain substances, such as radium, plutonium, and strontium, have a tendency to become a part of the chemical structure of bones and remain in the body for many years. It is with these substances—principally radium—that we have had most of our experience with chronic internal source radiation injury. The principal results are bone destruction and bone cancer occurring some 20 years after deposition of the radioactive substance. Anemia may occur within a few years after rather large amounts of radium have gained access to the body, but in lower doses—10 to 30 micrograms—it is not a prominent finding.

## Medical Societies.

### AUSTRALIAN PÆDIATRIC ASSOCIATION.

THE annual meeting of the Australian Pædiatric Association was held at Canberra on March 30 to April 1, 1957, Dr. T. Y. NELSON, the President, in the chair. Part of this report appeared in the issue of November 2, 1957.

#### Lung Cysts in Children.

J. M. ALEXANDER read a paper entitled "Cystic Disease of the Lungs in Childhood" (see page 676).

HOWARD WILLIAMS (Melbourne) said that clinicians were often faced with the problem of children with clinical evidence of pulmonary suppuration and radiological evidence of large cysts, frequently bilateral. When he examined tissue from such patients, the pathologist frequently had difficulty in deciding whether the cysts were of congenital origin or acquired, as infection had so altered the structure of lung tissue. Dr. Williams considered that most of the evidence indicated that the disease in the case presented was acquired, rather than congenital, but that it would be impossible to exclude some underlying congenital basis.

R. C. GODFREY (Perth) discussed lung cysts in infancy. He said that it had recently been appreciated that in the past many such cysts were labelled as congenital simply because they had appeared in early life, or, having been removed surgically or examined *post mortem*, they were described as congenital because of their histological characteristics. Those criteria had been criticized by Caffey and Conway, and Caffey had shown that the appearance of the lining of the so-called congenital cyst, with its epithelial layer and fragments of cartilage and muscle, was by no means pathognomonic, because all those features might occur in an acquired cyst as it enlarged. Moreover, congenital cysts should be as common in newborn and stillborn infants as in older children; yet Caffey was unable to find a single case in the examination of 5000 neonatal chest skiagrams. He quoted the experience of the Babies' Hospital in New York, where in 22 years only one case was found in a stillborn infant, and of Potter, who had found only two. True congenital cysts were certainly rare, and it was probable that whatever their aetiology, air-containing lung cysts occurred or were maintained because of a check-valve mechanism in their bronchial connexion.

Dr. Godfrey said that in the rare true congenital cysts there had been described a congenital anomaly of the bronchus which could produce cystic dilatation after birth as a result of a check-valve mechanism. The acquired lung cyst was probably produced during a pathological process within the lung. There was little doubt that staphylococcal pneumonia was the commonest cause of lung cysts in infancy. Of 44 cases of staphylococcal pneumonia reported from the Princess Margaret Hospital for Children, Perth, in no fewer than 29 was there radiological evidence of lung cysts at some stage of the patients' illness. In a review of his own cases of lung cysts, he had been able to find only six that were not certainly associated with staphylococcal pneumonia. Three of those patients were asthmatics. One developed a balloon cyst during an acute respiratory infection of uncertain nature, one was a migrant whose

previous history was unknown, and one was suffering from congenital lobar emphysema. Lung cysts had been reported in tuberculosis and even in paraffin pneumonia, and it was likely that, in those, too, it was a bronchial check valve that produced the cyst. Because of those observations the following classification was suggested:

#### 1. True congenital cysts:

Fluid-filled paratracheal cysts.

Fluid-filled cysts due to lymphangiomatous malformation.

Cysts in sequestered lobes.

#### 2. Ball-valve cysts:

(i) Resulting from developmental lung defects—(a) forming after rupture of a fluid-containing cyst into bronchus, (b) associated with bronchial malformations, (c) congenital lobar emphysema.

(ii) Resulting from acquired lung disease—(a) in staphylococcal pneumonia, (b) in other pneumonias including paraffin ingestion, (c) in asthma bronchitis (ball-valve—due to bronchial swelling), (d) in tuberculosis (ball-valve—due to bronchial deformity), (e) in trauma (ball-valve—due to bronchial injury).

Dr. Godfrey said that in type 2 (i), symptoms were likely to occur at or very shortly after birth, whereas in type 2 (ii) they would develop later. It might be argued that congenital lobar emphysema should be excluded from the classification, on the grounds that it was not a true cyst. However, in those cases Fischer and others had described developmental anomalies of the bronchial structure of the affected lobe, and it might well be that the emphysema, and the failure of the lobe to collapse when removed from the body, were due to check-valve mechanisms, acting uniformly throughout the finer air passages. The condition would then be analogous to emphysema occurring in chronic bronchitis in adults—probably associated with obstruction to expiration from the finer air passages.

Discussing treatment, Dr. Godfrey said that if the very great majority of lung cysts were acquired, were potentially reversible and tended to regress spontaneously, surgical excision in those cases would appear to be contraindicated; a follow-up investigation of his cases showed that at least all cysts occurring in staphylococcal pneumonia had eventually disappeared. Unfortunately, rapid enlargement of a lung cyst might produce respiratory embarrassment which required immediate relief, and for those patients radical surgery was usually recommended. During the last three years a number of infants with tension cysts arising during staphylococcal pneumonia had been successfully treated by stab thoracotomy and the insertion of a catheter into the cyst. That treatment effectively relieved respiratory embarrassment and usually led to prompt disappearance of the cyst, eventually allowing the lung to become clinically and radiologically normal. Because of the success obtained by that method in staphylococcal pneumonia, they argued that it might be used as a first line of treatment in all cases in which rapidly enlarging cysts were causing respiratory embarrassment.

E. S. STUCKEY (Sydney) pointed out that treatment in the case presented had been conservative for five years, and that marked improvement had then followed surgery. Despite the fact that there had been no evidence of cysts in the first X-ray films, he still considered that the disease was due to congenital anomalies which allowed the cysts to expand. However, the problem of aetiology in that case was not yet solved.

KATH CAMPBELL (Melbourne) described one case in which severe dyspnoea had been present from birth. X-ray examination of the patient at the age of two days revealed a large cyst in the upper lobe of the left lung. At operation at the age of six weeks, a large cyst was seen to occupy the whole of the upper lobe. The lower lobe was fibrous and contained a number of small cysts. The patient had progressed satisfactorily for several years, but the abnormality in the lung still remained. Dr. Campbell said that the case was an instance of a congenital lung cyst.

Dr. Godfrey, in reply, suggested that the primary defect lay in the bronchus, which allowed cystic changes to develop in the rest of the lung; but even those cases were rare.

### Disorders of Reading in Childhood.

#### Reading Disorders in Childhood.

MISS E. KEMP (Sydney) read a paper entitled "Reading Disorders in Childhood" (see page 678).

#### Neurological Aspects.

LEONARD RAIL (Sydney) read a paper entitled "Neurological Aspects of Reading Disorders in Childhood". He said that Miss Kemp had considered three types of children who suffered from reading disability: (a) the backward child, (b) the brain-damaged child, (c) the child suffering from a specific reading disorder. He did not propose to consider the first group of backwardness from a neurological point of view. The problem of detection was a matter of clinical assessment with the help of intelligence tests and investigations for cerebral disease. There was also a group of essentially optical defects which he would not consider further. The other two groups—brain damage and so-called specific disability—could be dealt with together. It was probably true to say that one was never dealing with a pure reading disability. As Critchley stated, if adequate examination was made there was always some defect in the field of language, and not infrequently abnormalities were present which could be ascribed to parietal lobe defect. Also, it became clear that in affected children there were frequently associated abnormalities, such as speech disorders, emotional immaturity and often undue clumsiness or poor motor coordination. In the brain-damaged group there might be other disabilities such as hemiplegia and, of course, mental defect. The lesion which was known to cause alexia was damage to the angular gyrus region, the meeting place of the parietal, temporal and occipital lobes. However, that applied mainly to acute damage, and in those cases it was often associated with aphasia and other symptoms, such as disturbance of right-left orientation and other defects of the body scheme.

Dr. Rail went on to say that Henschelwood had proposed that reading disorder was due to an agenesis of the angular gyrus. However, in Krynaus's cases of hemispherectomy (with right hemiplegia), the left hemisphere was completely removed in ten cases without lasting aphasia or dyslexia. It was, therefore, doubtful whether agenesis of one small area such as the angular gyrus could give rise to lasting dyslexia. For the same reason Gesell and Amatruda's statement that minor and unrecognized brain damage at birth might lead to reading and speech disorders at a later date could be seriously doubted. Dr. Rail thought that unless a physiological disturbance such as persistent dysrhythmia of an epileptic type was present in the damaged areas, speech and reading disability was unlikely to be present because of compensating mechanisms. Extensive damage to both hemispheres could, of course, interfere with the development of the reading functions; but there mental backwardness would most likely accompany the syndrome. In the vast majority of cases of dyslexia, no brain damage had been suspected or found by any of the usual methods. In that group one had to look elsewhere. In a large number of such cases a familial element was present. Hollgren in 1950 had shown that reading disability was inherited as a dominant, non-sex-linked, single gene. Thus the causal mechanism must account for (a) heredity, (b) poor motor coordination, (c) dyslexia, (d) speech disorders, such as stammering and hesitancy, and (e) emotional instability. Disturbances of dominance had been postulated as the cause by many authors. However, to summarize the position in the words of Brain: "Anomaly of handedness is a symptom and not the cause of the disorder underlying the congenital aphasias." Thus disturbed dominance might be added to the symptoms. At that stage it should be noted that all those symptoms and signs were normal for a child aged below two or three years—in other words, at an earlier stage of development. Much work had appeared in recent years on failure of maturation of the temporal lobe and its relation to emotional stability. A similar concept of delayed maturation had recently been applied by Drew to the problem of congenital dyslexia. He suggested that the condition might be due to immaturity of the parietal lobe structures. Dr. Rail thought that if a maturation defect was to be postulated, it should be applied to a wider field than the parietal lobe, including areas of the temporal and possibly the occipital lobe. That was an attractive hypothesis. Such a condition could be hereditary, and it would account for the failure to develop satisfactory motor coordination, the failure to acquire control of speech. The associated emotional immaturity, often ascribed to psychological causes, might be due to temporal lobe immaturity. It would also account for the failure to develop normal dominance, which was, of

course, a more specialized motor control. Finally, it could account for the failure to correlate the more difficult and abstract types of visual stimuli, which were needed to allow reading. Such a defect was also compatible with recovery, which normally occurred in a considerable proportion of cases—an analogous situation to the improvement in emotional control which occurred in temporal lobe maturation problems. The multiple factors involved in what might appear to be a simple defect of learning underlined the necessity for cooperation between the physician and the educationalist, with the help of neurological, psychiatric and ophthalmological services.

T. Y. NELSON (Sydney) thanked Miss Kemp for making the trip to present her paper and for drawing attention to the problems. He said that for people entrusted with those patients it was difficult to know what treatment should be carried out, and Miss Kemp's remarks had been helpful.

### The Management of Double Ureters in Childhood.

D. STEPHENS (Melbourne) read a paper on "The Management of Double Ureters in Childhood" (see page 679).

### Congenital Syphilis: A Retrospect.

ROBERT SOUTHEY (Melbourne) read a paper on congenital syphilis. He began by quoting a statement by Oliver Wendell Holmes: "Every man is an omnibus in which all his ancestors are seated." Dr. Southey then said that he proposed to take those present back a little over a third of a century—35 years, in fact. The scene was the special clinic, which had been established by his friend and colleague, Howard Boyd Graham, for the purpose of supervising the treatment of children suffering from syphilis and gonorrhoea and of following up their after-care. In those days congenital syphilis was a definite clinical entity, frequently seen, and necessitating a separate ward for the care of the patients. The therapeutic armamentarium consisted of iodides, mercury, arsenic and bismuth. It was generally accepted that arsenic was the most efficient preparation, and bismuth was coming into fashion. For infants and children who would necessarily require a long course of treatment, the ideal aim was to provide an injection which would be therapeutically effective and at the same time be accompanied by a minimum of pain and discomfort, and be free of local or general reaction. "Salvarsan" (606) and "Novarsenobillon" (914) were the preparations of choice for adults, and when they could be administered intravenously they fulfilled most of those requirements. For little children and babies it was a totally different proposition, as the injections generally had to be given by the intramuscular route. In an endeavour to make that possible, "Novarsenobillon" was given in a solution of glucose and gualacol, the former with the idea of aiding absorption, and the latter in an attempt to relieve local discomfort. That technique was not entirely successful, and it only required one or two severe local reactions after injection to discourage a mother from attending for further treatment for her child. Then a French preparation, "Sulfarsenol", had entered the field, and that was the answer to the paediatrician's prayer, for the precious yellow powder could be dissolved in sterile distilled water and then administered by the intramuscular route or the deep subcutaneous route (beneath the *tensor fasciae latae* on the lateral aspect of the thigh), and it was found to be readily absorbed without local or general reaction. Furthermore, it was most effective in producing a dramatic clinical response and an equally gratifying serological improvement. Then followed the bismuth preparations, the most effective and satisfactory of which were suspensions of bismuth hydroxide in oil—"Muthanol" (French) and "Bismol" (Australian). Those injections also were free of local or general reaction, and the response was satisfactory. Many children were treated with bismuth alone, and the results were equally encouraging. An idea of the size of the clinic might be gained when it was recalled that once a week 20 to 25 children were given their injections, and anything from 10 to 15 had blood specimens collected for the serological tests. Those tests were carried out by the quantitative technique at the Walter and Eliza Hall Institute of Medical Research at the Melbourne Hospital. It was necessary to provide the laboratory with samples of serum and a summary of the clinical history and progress reports, and that alone involved a considerable amount of time and energy at the end of an already long and strenuous session. Miss F. Eleanor Williams had carried out those tests in a highly efficient manner, and her reports were most encouraging and helpful in judging the progress of any particular child under treatment. The result was expressed in the number of minimum haemolytic doses of complement fixed by the particular specimen of serum.

Dr. Southey went on to say that the mothers naturally were deeply concerned about their children, and frequently asked questions such as the following: "Will the treatment be effective?" "Will Mary recover from this disease, and will it be possible for her later to marry and have healthy children?" "Will Billy be cured and can he become a healthy citizen eventually?" Workers at the clinic, with their faith in their therapy, had perforce to maintain a constant optimism, and assure the distracted mothers that eventually all would be well if they would be patient and persevere with their regular attendance for the children's treatment. Without exception, of course, the mothers had to be treated also, and that meant for each mother at least two visits a week, or frequently more, between two different hospitals.

Dr. Southey said that the florid clinical signs literally melted away under the treatment, as did the radiological manifestations of periosteal and bony changes. In parallel with that was the steady improvement in the serological reports which were given quantitatively. That method was a great improvement on the older reports, which repeatedly showed "strongly positive", but with no indication as to whether the reaction was becoming less strongly positive with repeated courses of injections. It was particularly useful also in indicating an optimum response to either arsenic or bismuth on the part of any individual child. In due course the great majority of the patients showed a very satisfactory response, and finally the eagerly awaited "negative" report came to hand—with the one exception of the patients suffering from nervous system involvement with a positive reaction in the cerebro-spinal fluid. Those reports remained obstinately "positive", and the condition resisted all treatment, including malarial therapy; eventually the patients succumbed to the disease, often before adolescence. With the great majority of the other children, once the "negative" report appeared and a further one or two courses of injections had been continued, the serological reaction usually remained "negative". The finding was checked each year until the age of fourteen years (the age limit for the hospital). Cerebro-spinal fluid was tested also, and a negative result confirmed the cessation of active treatment. Dr. Southey said that as the years had passed, it had been his privilege, and a great joy, to maintain contact with a considerable number of those patients, and even to see them bringing their own children along for a general examination and a blood test. Again the late results had been most encouraging, and almost every one of the patients so followed—male or female—had maintained the improvement. The girls had married and passed through the acid test of pregnancies. They had remained well, and their children had been well clinically, free of stigmata and with negative serological findings. The boys also had remained well, and many had married. There had been no instance of infection of their wives, and their children had been quite healthy. Dr. Southey described in detail the clinical history of a female infant, who had presented in 1924 at the age of six months; she manifested most of the stigmata of florid congenital syphilis, particularly extensive bony involvement, and the serological findings were strongly positive. She had been given appropriate treatment over the years, and in 1957 she was well, and was married, with three healthy children. Dr. Southey thought that was surely the final confirmation of the efficacy of her treatment as an infant. In conclusion, he summarized his retrospect on congenital syphilis in four points: (i) Arsenical and bismuth treatment had been proved effective and lasting over a long period. (ii) There had been an opportunity of observing a disease throughout its whole course. (iii) A particularly gratifying end result was illustrated by the cured patient who produced healthy children. (iv) The clinic staff's optimistic answers to the questions of the anxious mothers were vindicated. (v) The story provided ample confirmation of the old adage: "All's well that ends well."

T. Y. NELSON (Sydney) said that it was most interesting to hear of that follow-up series. He asked Dr. Southey whether any subjects of congenital syphilis were seen now.

Dr. Southey replied that such cases were indeed uncommon, and it was quite usual not to see such a patient for six to twelve months. That was an excellent example of preventive medicine—the infant being treated before it was born.

FELIX ARDEN (Brisbane) asked whether Dr. Southey had seen pink disease develop in a child treated with mercurial injection.

Dr. Southey replied that he had used that treatment in hundreds of cases and had not seen a case of pink disease result.

### Palmar Pulsation in Patent Ductus Arteriosus.

D. STUCKEY (Sydney) read a paper entitled "Palmar Pulsation: A Physical Sign of Patent Ductus Arteriosus in Infancy" (see page 681).

D. G. HAMILTON (Sydney) asked Dr. Stuckey whether he considered that an excess of palmar subcutaneous fat in childhood might give rise to a false positive sign.

Dr. Stuckey replied that he did not think that that would occur, as palmar subcutaneous fat was not pronounced at any stage in life.

D. H. COHEN (Sydney) said that that sign had been very helpful in the diagnosis of difficult cases in young children.

### Hermaphroditism.

#### Ambisexual Development.

HOWARD WILLIAMS (Melbourne) read a paper on ambisexual development. He said that from time to time children were born with external genitalia, genital ducts and even gonads which had both male and female characteristics. Children with such ambisexual features had been called hermaphrodites, the word hermaphrodite being derived from Greek mythology. Hermaphrodite, the son of Hermes and Aphrodite, was described as a womanish young man with breasts and long hair. Considerable confusion and serious mistakes had frequently occurred in the diagnosis and management of patients with ambisexual development, owing to ignorance of the various types, and also resulting from failure to appreciate that masculinity and femininity depended on other factors than gonadal hormones. Wilkins and his associates had recently shown that psycho-social factors were of the greatest importance in determining male or female behaviour and attitudes.

Dr. Williams said that there were two principal groups of patients exhibiting ambisexual features: (i) those in whom active secretion of a hormone caused sex reversal; (ii) those in whom genetic mutations or intrauterine factors had acted during development of the fetus. In the hormonal sex reversal group, by far the commonest type were females who were virilized by androgen secretion from the adrenal glands. Those androgens were an abnormal metabolic product due to an enzyme deficiency, so that cortisone could not be synthesized. Such patients were commonly referred to as affected by pseudo-hermaphroditism due to congenital adrenal hyperplasia. In the second group, in which the causal factors were probably either genetic mutations or intrauterine factors, three outstanding characteristics were observed. First, there was a wide diversity of abnormalities of the external genitalia, uro-genital sinus and genital ducts. Secondly, the gonads could be testes, or ovotestes, or ovary and testis, or ovaries. Thirdly, secondary sex characteristics did not develop until puberty, and it was impossible to predict whether the subjects would be male or female in character.

Dr. Williams further said that three main classes of infants required full investigation, and such investigations should be undertaken early before any pronouncement was made concerning sex. The first was the group with a uro-genital sinus with varying degrees of labial fusion and enlarged clitoris or abnormal phallus. The second comprised patients with cryptorchidism and either hypospadias or an abnormally developed penis. The third consisted of patients with female external genitalia but palpable gonads in both labia. It was obvious that older infants or children who developed ambisexual features required full investigation. One of the most important investigations was to determine whether there was an active hormonal basis for the abnormalities. Quantitative analysis of a 24 hour specimen of urine for excretion of 17-ketosteroids was the most important single estimation. Figures of over one milligramme per 24 hours' specimen were usually abnormal in infants; but occasionally a normal infant did excrete as much as one milligramme. Pregnanediol estimations were particularly valuable in the investigation of patients with congenital adrenal hyperplasia; but the biochemical analysis of small amounts of that hormone was very difficult. Raised ketosteroid and pregnanediol excretions were strong proof of congenital adrenal hyperplasia. The next step in investigation was radiographic study by injection of a radio-opaque substance into the uro-genital sinus or urethra, to demonstrate the urethra and to determine whether there was a vaginal communication. Urethroscopic examination might be used as an additional investigation. Nuclear sexing by examination of mouth scrapings or skin biopsy should be undertaken. If the patient had a uro-genital sinus and enlarged phallic structure, an increased urinary secretion of 17-ketosteroids and pregnanediol, a vagina and a female nuclear sex pattern, then congenital adrenal

hyperplasia could be confidently diagnosed and the infant treated with cortisone. In the non-hormonal group it was usually necessary to carry out a laparotomy to determine accurately the type of gonad and genital duct system present. Only after all the evidence from the different sources was available could the most sensible sex of rearing be determined. The type of external genitalia was most important in influencing the decision of sex. If it was impossible for the surgeon to construct a penis and there was a vagina present, then it was advisable to rear the infant as a girl, even if the gonads were testes or a testis and ovary were present. Each case must be decided on its merits, and no one factor such as the type of gonad or nuclear sex pattern should be all-important in deciding the sex in which the child should be reared.

Dr. Williams finally stressed three points in management. He said that, first, a clear, definite opinion on the sex of the child must be given to the parents—"your baby is a boy", or "your baby is a girl". No hint of any doubt was justifiable. Secondly, the parents must be assured that the child would grow up as a normal girl or boy and that he or she would not develop any bisexual features or have any abnormal psychological traits as sexual perversion. That particular feature often worried parents, who had gleaned inaccurate information from popular magazines. Thirdly, the surgeon should construct, if necessary, normal genitalia for the particular sex at as early a date as possible. Normality of external genitalia was most important in stressing the normal sex to the parents. Any child with ambisexual features should be referred to a physician and surgeon who were well versed in the diagnosis and treatment of such abnormalities. Close collaboration of those two was essential for the best management of such patients.

#### Diagnosis and Management of Hermaphroditism.

F. DOUGLAS STEPHENS (Melbourne) discussed the diagnosis and management of hermaphroditism. He said that surgery entered into the management of those children for investigations conducted from below and above, and for correcting the external deformities of the genitalia. Several investigations were conducted from below. Micturition cystourethrography was employed to outline the bladder and urethra and to discover whether reflux occurred into the vaginal lumen during micturition. Retrograde injection of iodine through the external opening of the uro-genital sinus might be necessary to display the vagina, if it was not defined by the micturition method. Sometimes the uterine lumen and even the lumina of the tubes were visualized by radiography. If the presence of a vagina was suspected, but no vagina was demonstrated by either of those techniques, urethroscopy and direct catheterization of the vaginal orifice under vision were required. Reflux was of little importance, except in those older children who had been brought up, in error, as hypospadiac males. After reconstruction of the pendulous urethra, reflux into the vagina might then lead to urinary stagnation, infection and deposits of calcareous material. Those investigations concerned the identification of genital organs; but laparotomy for that purpose was obligatory for certain children, particularly those with doubtful external genitalia and normal ketosteroid excretion. Small pieces of all gonads should be taken for microscopic examination of sections.

Dr. Stephens then discussed the correction of the external genital anomalies. He said that clitorrectomy and vaginoplasty might be required to effect a feminine appearance. Total or near-total removal of the *corpora cavernosa* had been carried out in his cases. The operation had been performed, for preference, in the first few months of life. Vaginoplasty was performed at the same time, when the vagina opened into the urethra at the usual very low site. Reconstruction of the pendulous urethra in the child to be brought up as a male should be carried out in the pre-school days. The Denis Browne technique of repair of hypospadias had been used for such children. Dr. Stephens finally said that surgeons should suspect the sex of hypospadiac patients when one or both testes were undescended. Ketosteroid estimations, skin biopsy, investigations such as urethrography, urethroscopy, pyelography and even laparotomy were necessary to obtain all available material and data to assist in the proper determination of the patient's sex.

#### Discussion.

R. H. VINNS (Sydney), in opening the discussion, said that he could do little more than underline some of the things which had been said. However, he felt that he must make some objection to the clarity of ideas on sex differentiation in Dr. Howard Williams's paper. Five years earlier his own ideas on sex, with merely Freudian reservations, had been

clear cut and simple: if the gonads were testes, the person was male; if they were ovaries, the person was female. Now it was known that testes might produce oestrogens and so be responsible for breast development and the appearance of other feminine characteristics at puberty in the externally feminized male—pseudohermaphrodite. The ancient statues of Hermaphrodite represented a figure having breasts and feminine bodily contours, together with male genitalia. Living replicas of those statues were given the euphonious name of Klnefelter-Reifenstein-Albright syndrome, and Barr's technique of nuclear sexing had shown that at least some such people had a feminine type of nuclear chromatin pattern. Therefore, reliance could not be placed on the gonads in assigning sex. As far as the paediatrician was concerned, most problems involving sex determination presented in the first months of life. In approaching the diagnosis of sex in an infant at that age, the family history was of some importance. A history of early masculinization or precocity or death in infancy all suggested the possibility of congenital adrenal hyperplasia. As far as clinical examination was concerned, the importance of not making a diagnosis purely on the basis of the approximation of the genitalia to the normal male or female pattern could not be stressed too much. However, it was true that careful examination might help. For instance, if a gonad was palpable externally, the infant was not a female. In that small group of children with multiple congenital abnormalities and hypertrophy of the clitoral prepuce which he himself had described in 1956, diagnosis could be hazarded on careful clinical examination alone. The failure of normal weight gain to occur in the first weeks of life should make one suspect that adrenal hyperplasia of the salt-losing type was responsible for a genital abnormality.

Dr. Vines went on to say that in nine of the eighteen infant girls with adrenal hyperplasia whom he had examined in the last five years, that complication had been present. Nuclear sexing in experienced hands was always worth doing, and estimation of the 24-hour 17-ketosteroid excretion was also desirable on at least one occasion for the child in regard to whose sex a doubt had arisen. Dr. Vines said that he had not found pregnanediol examination to be a reliable indicator of the presence of adrenal hyperplasia. Urethroscopy and radiological techniques using radio-opaque dyes were of assistance to the surgeon in planning plastic procedures, but were of little value in making a diagnosis. Gonadal biopsy might sometimes be necessary, but he considered that the necessity was largely experienced by the doctor anxious to make an academically correct diagnosis, rather than imposed by the requirements of the patient. When doubt remained after appropriate investigation as to the better sex in which to rear the child, the genital anatomy was the most important factor in assigning the gender role. In other words, if a serviceable phallus was present, a male gender role was usually best. It was most important that a dogmatic statement be made to the parents as to the sex of the child, and that it should be made without undue delay; for it had become more and more apparent that it was not the type of gonads or the type of nuclear chromatin present that determined the sexual attitudes of people, but the gender role in which they had been reared in the first two or three years of life.

PETER JONES (Melbourne) asked the maximum age at which a change of gender sex could be attempted with hope of success.

Dr. Williams replied that his experience was limited, but that the problem was difficult. Certainly children aged less than one year, and probably children aged less than two years, would respond satisfactorily to a change in gender sex. The group of workers from Baltimore had suggested that the sex of infants aged less than two years could be changed, and that children aged more than four years should be left; but they did not make recommendations for children aged from two to four years.

PROFESSOR ASHLEY WEECH (Cincinnati) asked what psychological measures were taken to prepare those children, who were being brought up as males, for a rapid growth spurt giving them physical superiority over their fellows, followed by eventual stunting and physical inferiority.

Dr. Williams replied that cortisone was being given to two patients who were being reared as males to retard the growth spurt. He thought that the home environment was most important in preparing the child for those changes.

PROFESSOR LORIMER DODS (Sydney) said that he usually explained to the parents of children who were being brought up as girls that the development of the external genitalia was not completed, but that otherwise the children were normal girls, and that the development of the genitalia would be completed later, with medical help.

H. N. B. WETTENHALL (Melbourne) said that Dr. Lawson Wilkins had emphasized the importance of examination of the external genitals in making the decision as to whether to change the gender sex. Dr. John Monery, the psychologist associated with Dr. Wilkins, said that there was psychological difficulty in children when a decision to change the child's sex after the age of four years was carried out. Dr. Wettenhall had seen several children in Dr. Wilkins's clinic in whom psychological difficulties had occurred.

#### The Pierre Robin Syndrome.

J. STEIGRAD (Sydney) read a paper entitled "The Pierre Robin Syndrome" (see page 682).

KATE CAMPBELL (Melbourne) said that she had noticed an increase in such cases recently. The possible aetiology was interesting. In one of her cases the mother had had a cleft palate. Another affected child had been born into a district where there had been an epidemic of infective hepatitis. There had been an increase of congenital abnormalities in that district. Nursing of patients was difficult and the results of surgery were disappointing. After the Douglas operation, sutures frequently were torn out. Tracheotomy had been necessary in several cases. There was a risk of sudden death if tracheotomy was not performed in those cases.

ROBERT SOUTHEY (Melbourne) said he considered that there might be a familial tendency. He had seen two families, in each of which two children had been affected. They had died suddenly. Tracheotomy should be performed at an early stage if feeding difficulties were present.

PROFESSOR ASHLEY WEECH (Cincinnati) agreed that surgery was often inadequate, in that sutures were frequently torn out. In the hands of an experienced surgeon the Douglas procedure was better; tracheotomy was better if the surgeon was not familiar with the Douglas operation.

#### The Ectopic Ureter.

E. STUCKEY (Sydney) read a paper entitled "The Ectopic Ureter" (see page 688).

DOUGLAS COHEN (Sydney) pointed out that the sign of dissociated micturition was very helpful, and that he considered that intravenous pyelography should also be carried out, as lateral displacement in the pyelogram by the ectopic ureter was often noted. Insertion of indigo-carmin dye into the bladder followed by the passage of colourless urine from the ectopic opening was also a valuable sign.

DOUGLAS STEPHENS (Melbourne) said that the dye test had not been used in his unit. He had never before seen a case of ectopic ureter in which the ureter opened high in the vagina. In his experience the ectopic ureter always accompanied the orthotopic ureter through the bladder wall into the submucous space of the bladder and thence to its final orifice in urethra or vulva.

Dr. Stuckey, in reply, explained that he considered that the cystic protrusion was probably the dilated lower end of the ectopic ureter, which was subsequently incised, with the result that the opening became higher in the vagina. He could not determine the original site of opening of the ectopic ureter.

#### A Diencephalic Syndrome.

PROFESSOR LORIMER DODS (Sydney) read a paper entitled "A Diencephalic Syndrome of Early Infancy" (see page 689).

T. Y. NELSON (Sydney) asked whether Russell's cases were associated with tumours.

Professor Dods replied that Russell had indicated by letter that that was so.

J. COLERATCH (Melbourne) asked whether any of the other patients had had hypertrophic pyloric stenosis.

Professor Dods replied that they had not.

D. MCKAY (Brisbane) asked whether the case of hypertrophic pyloric stenosis was a true case with typical appearances in sections.

Professor Dods, in reply, said he regretted that he had no direct evidence, but that a report from another hospital had stated that the sections were typical.

PROFESSOR ASHLEY WEECH (Cincinnati) said he believed that the condition was a syndrome. He had seen a patient with oligodendroglioma of the diencephalon who had died within 24 hours; examination of the cerebro-spinal fluid had shown a gross decrease in glucose. Another patient, a child aged 14 months, had presented a marked cracked-pot percussion note. Later obstructive signs appeared. Professor Weech

asked Professor Dods whether any observation of McEwan's sign had been made in his cases.

Professor Dods replied that there was no record of that observation.

Dr. Nelson said that he had found that sign an unreliable one until late, when there might be widely separated sutures.

M. SOFER SCHREIBER (Sydney) said that he considered the condition to be a syndrome. He asked Professor Dods whether there had been a raised protein content in the cerebro-spinal fluid.

Professor Dods replied that, in three cases, the protein content was raised at a late stage. In one case it was raised early.

#### Hepatitis in Infancy.

FELIX ARDEN (Brisbane) read a paper entitled "Hepatitis in Infancy" (see page 683).

H. RISCHBIETH (Adelaide) described a child, aged six weeks, who had had persistent jaundice from birth, and passed very pale motions and dark urine. Examination of eight daily specimens revealed no stercobilin in any. The serum bilirubin content was 18 milligrammes per 100 millilitres; otherwise liver function tests gave essentially normal results. Laparotomy revealed only a thread-like remnant of the hepatic duct communicating with the liver. Injection of "Diodrast" into the gall-bladder failed to demonstrate a lumen in the duct. No anastomosis was performed. Liver biopsy revealed signs of obstructive jaundice. Ten days after laparotomy the stools became darker and stercobilin was detected. Since then there had been a progressive diminution in and finally complete disappearance of jaundice. The child now appeared perfectly well, and seemed to represent a recovery from infantile hepatitis.

Dr. Arden said that he could not explain the reappearance of stercobilin in the stool in that case in the apparent absence of a patent hepatic duct.

HOWARD WILLIAMS (Melbourne) said that the problem in a jaundiced neonatal infant was to decide whether the abdomen should be opened. In general, liver function tests did not help. The most helpful investigation was examination of the stool for the presence of bile. The stool should be collected by introducing a finger into the rectum, as stool collected after it had been passed might be contaminated with urine. If stercobilin was absent from the stool over a period of one month, exploratory laparotomy should probably be performed.

#### The Decline in Breast Feeding.

CLIFTON WALKER (Sydney) discussed the decline in breast feeding. He said that many causes might operate to make nursing difficult. Mothers noted the excellent progress made by artificially fed babies, and they knew exactly how much milk the infant received at each feed. A strong sucking stimulus from the baby was necessary to ensure an adequate supply of breast milk, and the lethargic type of infant was a cause of real difficulty. Even when there was strong suction the "nervy" mother might fail, because, as Waller had stated, she could not "give" the milk—she was too tense and worried. Some mothers were unable to nurse their infants because they had to help their husbands at work, or were overwhelmed by home worries. Other mothers said that nursing spoilt their figures, and a few objected to it on the grounds that it was repulsive. Modern living conditions, which made it unavoidable for some families to occupy only one room, presented a considerable difficulty. Trouble was also caused by nipple abnormalities, accompanied by painful nursing and constant expression of milk. Some husbands objected to their wives' breast feeding the baby, and a few women frankly admitted that it interfered with their social life. An increasing number were disinclined to nurse their infants, and a few refused outright.

Dr. Walker went on to say that the essentials for breast feeding were a mother who was healthy, contented and with a will to nurse her baby, who had normal breasts and nipples, and who was feeding a healthy infant with adequate suction. Mothers needed to be helped over several hurdles. They were upset by the crying baby who appeared healthy, but suffered from colic for a number of reasons. When there was tension in the home, and perhaps the mother or father was "nervy", or they lived with "in-laws", a patient interview with both parents and the prescription of a sedative for the mother would often be effective. The gulping baby who took in much air at feeds would usually be transformed into a relatively peaceful infant by the administration of a sedative before his feeds; but the dosage

should be adequate. A rectal examination should be a routine procedure; occasionally the colic would be dramatically relieved by dilatation of a tight anal sphincter. Nipple difficulties were a frequent source of trouble; many could be averted by adequate ante-natal care. Many colicky babies were found in families in which there was a history of allergy. Orange juice seemed to be a common offender. There was hardly ever any indication for giving orange juice to the young breast-fed baby. The administration of sodium citrate appeared to make the colic worse by causing rapid evacuation of the unclotted milk from the stomach, with the production of hunger pain. Frequent dosing of the baby with fluid magnesia was apt to cause colicky pain; it was almost never necessary. The administration of galactogogues to the mother was of no avail; the intake of extra fluids did not result in an increased supply of milk. Dr. Walker advocated the use of the pacifier; he said that sucking on the "dummy" was not accompanied by swallowing movements, so that there was no intake of air. There was no need for the baby to be kept for ten minutes at each breast; many would do well with as little as five minutes at each breast. Forcing the baby to remain longer often resulted in a large intake of air, followed by much crying. When supplementary feeding became necessary, the test should be examined. If it was too difficult, the baby took too long to obtain a few ounces, the result being wind and much crying. Scales were a menace in the home; weekly, and later bi-weekly, weighing was sufficient. The use of pumps for stripping was inadvisable; Waller thought they should be "museum pieces". The baby's time at the breast should not be limited because he was gaining weight rapidly; he should be allowed to gain 10 to 12 ounces a week if he wished. Early mixed feeding at the age of about three months was well received by mothers, and did not appear to interfere with satisfactory lactation.

Dr. Walker finally said that the nursing mother, especially with her first baby, needed understanding and encouragement from her nurse and doctor. He referred to the decline in breast feeding that had been reported from many countries, and gave some figures from his own practice. Of the last 530 babies in his own series, 62% had been fully weaned at three months. Of the mothers, 53% of *primiparae* failed in breast feeding and 58% of *multiparae*. Thus, 62% of babies that had received various types of care at large metropolitan maternity hospitals, at suburban and country hospitals, and even at specialist nursing homes and centres, were completely weaned at three months. A number had been fully weaned when they came under his care, and a considerable number were in difficulties, and persistence with breast feeding looked hopeless. Dr. Walker said that some women who failed were extremely anxious to adopt the natural method. Even before they left hospital, many babies were already receiving supplementary artificial feedings; that was often the beginning of the end. On arrival at home the baby was soon weaned. Dr. Walker wondered whether it was possible that breast feeding had become difficult to establish satisfactorily in hospital because of some subtle neurological disorder resulting from the mild hypoxia caused by the excessive use of analgesics during labour now demanded by the modern mother. If mothers could be kept in hospital longer, until lactation was well established, the results would be better. That was the practice in British hospitals for mothers and babies. With the present high cost of hospital care, mothers could not stay long enough to establish lactation on a secure basis, even if beds were available for the purpose. It was often stated that a placid and contented mother was an essential for smooth nursing. Under modern living conditions there were few such mothers. Rooming-in and self-demand feeding seemed like a last desperate attempt to bolster up the natural method. It seemed that the infant must bow to the inevitable.

KATE CAMPBELL (Melbourne) considered that successful breast feeding was dependent on two factors, the first being the attitude of the community, and the second the hospital care of the mother and baby. Dr. Campbell said that the fault lay, not with the mother, but with the routine. Most mothers wanted to breast feed their babies, but were not helped sufficiently either by their obstetricians or by their paediatricians. Rooming-in and demand feeding were natural aids to breast feeding. The baby would suck well when he was hungry, and that would stimulate milk production. The success of breast feeding was determined in the first five days in hospital. It was inhibited by the presence of congested breasts or sore nipples, and constant observation of the breasts was essential. "Six o'clock colic" could be prevented by the lying down method of breast feeding.

KATHLEEN WINNING (Sydney) heartily agreed with Dr. Campbell and reiterated that women did not receive enough

help on the problem, from either their obstetricians or paediatricians.

BARBARA MEREDITH (Melbourne) said that 95% of mothers were breast feeding their babies on discharge from the Queen Victoria Hospital, Melbourne.

Dr. Walker replied that the Victorian figures were really no better than those of other centres. He agreed that obstetricians should advise mothers in regard to breast feeding. Many factors were involved, and the problem was a world-wide one. Demand feeding was not satisfactory; children and adults required regular feeding; why not the infant? Writers who advocated demand feeding all seemed to admit that the baby settled down to a regular four-hourly schedule after a few weeks. If he found out by trial and error that that was what he wanted, why should he not be started off with regular feedings?

#### Acute Haemolytic Anæmia and Acute Nephritis in Infants.

S. E. J. ROBERTSON (Sydney) read a paper entitled "Acute Haemolytic Anæmia with Acute Nephritis in Infants" (see page 686).

V. COLLINS (Melbourne) said that he had had a similar case, in which the platelet count had been reduced, and asked Dr. Robertson whether that had been noted in either of his cases.

Dr. Robertson replied that thrombocytopenia had not been observed in those two children.

JOHN COLEBATCH (Melbourne) thanked Dr. Robertson for drawing attention to what appeared to be acquired haemolytic anæmia in association with acute nephritis. He said that frank haemolytic anæmia was not often seen in acute nephritis, but it might be that it was more likely to occur when the disease affected infants as young as those described by Dr. Robertson. Dr. Colebatch mentioned the need for caution in considering reticulocytosis and normoblastosis as evidence of haemolytic anæmia, particularly in a disease such as acute nephritis, in which hemorrhage was likely to be present. However, in Dr. Robertson's cases there had also been an increase of bilirubin in the blood and of urobilinogen in the urine, and those increases might be presumed to indicate hemolysis, provided that the presence of even a subclinical degree of liver disease could be excluded. Haemolytic anæmia could also be suggested strongly by other signs, such as spherocytosis and the presence of haemolytic antibodies; but proof of a haemolytic mechanism should usually be based on the finding of an increase of stercobilinogen in the stools (a sign he found most useful and practicable), or of the presence of methæmalbumin in the plasma, or of a decreased red-cell survival time. Dr. Colebatch agreed with Dr. Robertson's hypothesis that those infants had apparently had acute haemolytic anæmia. With regard to the pathogenesis, he asked whether there had been any evidence of malnutrition or other ill health in the infants prior to their anæmic illness, whether the nature of the infection preceding the nephritis was known, and whether there was any evidence of allergy in their histories.

Dr. Robertson replied that he had looked for methæmalglobulin in the urine, but that that had not been found at the stage when the children were first examined by him. Their previous health had been excellent, and there was no evidence of allergy. The preceding upper respiratory tract infection was not serious in either case, but every member of the respective families had had a cold, so he assumed that the disease was infective in origin.

### Out of the Past.

*In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.*

#### REVIEW.

[From *The Journal of Anatomy and Physiology*, Volume XXI, 1937.]

THE Myology of the Limbs of *Dasyurus Viverrinus* by Alexander MacCormick, Demonstrator of Anatomy, University of Sydney, N.S.W.

That this paper is a valuable contribution to the comparative anatomy of the marsupials is evidenced by the fact of the University of Edinburgh having awarded a gold medal to its author on his presenting it as his thesis for graduation as Doctor of Medicine.

It is gratifying to note such a paper as the result of original work done in the yet embryonic Sydney Medical School. We may hope that in future, much, if not most, of the comparative study of the Australian mammals may be carried on by scientific effort in this part of the world. The special value of Dr. MacCormick's paper lies in its being an attempt at the accurate systematic description of a part of the muscular arrangements of *Dasyurus*—an animal which has not hitherto received such special attention—with a view to the recognition of the homologies of these structures. At the same time the descriptions are, to some extent, collated with similar researches on other marsupials, recorded by various observers, and thus the new facts are assigned their place in the body of knowledge relating to this order. Such a relation of the facts constitutes no small element in the value of the whole. The paper is mainly or entirely descriptive, there is no attempt at scientific generalization, but for this feature the author is deserving of our approbation, for, although hypothesis and generalisation are the crown of scientific work there can be no more disastrous result in biological or indeed in any natural science than that which issues from attempts at hasty induction, or raw generalization, from insufficient or ill-considered data, or from the launching of hypotheses which have not undergone the most stringent and testing comparison with a large body of ascertained and admitted fact.

The mode of statement is, as might perhaps be expected, essentially that of human descriptive anatomy, with such modifications as are rendered necessary by the difference between the respective points of views of zootomy and anthropotomy. These modifications have apparently been reduced to a minimum and we detect in a few instances a certain lapse from absolute accuracy of statement due to the intrusion of the anthropotomical standpoint in the use of such terms as "anterior", "inferior" etc. Such errors are however but trifling slips and on the whole the descriptions are thoroughly accurate and admirably worded. In any case we welcome every such addition to the mass of ascertained and recorded morphological fact and not the less heartily since it is in this instance a record of the careful observations of an able worker in the University of Sydney.

### Correspondence.

#### THE BRITISH ROYAL COMMISSION ON LAW RELATING TO MENTAL ILLNESS AND MENTAL DEFICIENCY, 1954-1957.

SIR: I was most interested in the very excellent Special Article by Dr. J. E. Cawte in the Journal of September 28, but I cannot agree with his conclusion that adoption of the recommendations will be an expensive luxury.

The main recommendations of the Report of the Royal Commission<sup>1</sup> are: (i) that the *Lunacy and Mental Deficiency Acts* be repealed and be replaced by a single Act, which will provide for three groups of patient, (a) the mentally ill, (b) the severely subnormal, and (c) the psychopathic; (ii) that similar procedures should apply in each group; (iii) that care should be without legal formality unless compulsory powers are really necessary; (iv) that new procedures should be used when compulsory powers are necessary; (v) that any hospital should be able to admit (under compulsory powers or informally) any patient for whom it can adequately provide, but no hospital should be forced to accept patients for whom it cannot or does not make adequate provision.

It is pointed out that most mental defectives already under care would fall into the "psychopathic" group (feeble-minded psychopaths), and that this group would also include antisocial psychopaths who need segregation (at present housed in mental deficiency institutions and gaols).

It is emphasized throughout that compulsory powers should not be used unless absolutely necessary. I cannot see that the changes in the law recommended will necessarily add to the present cost to the community of the care

<sup>1</sup>Report of the Royal Commission on the Law Relating to Mental Illness and Mental Deficiency, 1957, H. M. Stationery Office, London.

of the sick and the infirm, nor do I believe that their application to the Australian scene would cause such an increased cost.

In most Australian States (all, I think, except Tasmania), there is no special provision for the care of feeble-minded and other psychopaths. But those who require segregation are segregated—in mental hospitals and gaols. I do not think that offering this class of person voluntary treatment will increase the number under care. But the principle of offering informal admission will, I am sure, result in a marked reduction in the number of mentally infirm and ill people who have now to be certified.

The recommendations envisage the abolition of the legal distinctions between mental hospitals and other special and general hospitals.

I have read the voluminous Minutes of Evidence given to the Royal Commission<sup>1</sup> and have been very impressed with the searching and pertinent cross-examinations of the witnesses. The Commission had the best available evidence to go on, and its Report is, I believe, a masterpiece of common sense based on sound reasoning from the evidence.

Differences of opinion will, and should, occur; but, as always, I expect that the most dogmatic criticism will come from those who are least informed. Those who are interested in this matter should read not only the Report but the Minutes of Evidence which are even more interesting and informative.

20 Murray Street,  
Hobart,  
Tasmania.  
October 10, 1957.

Yours, etc.,  
J. R. V. FOXTON.

#### THE MANAGEMENT OF RINGWORM OF THE SCALP

SIR: I was interested in Dr. Sharp's review under the above heading in the Journal of October 5, but was surprised to find no mention of the value of dihydroxy-anthranel ("Cignolin", "Dithranol" or "Anthralin") in the treatment of the malady.

Ever since the publication of my communication on "The Mycotic Flora of Surfer's Foot" (M. J. AUSTRALIA, March 18, 1939) I have been using this derivative of chrysarobin with very satisfactory results on all intractable mycotic infections, including ringworm of the scalp.

The drug must be used with caution. A 1% solution in pure benzole is carefully rubbed into a very small spot, and the patient told to report back the following day. Very occasionally the application causes blistering; if this has not happened, the solution is firmly rubbed in over and around the infected area. The area is treated in the same way three days later, and thereafter once a week for five or six weeks. In my experience it has never been necessary to shave or epilate when using this application. I believe the high penetrating power of the benzole solution is a very big contributing factor to the efficacy of this fungicide.

In conclusion, I have never known the drug to cause blistering on hairless areas.

Yours, etc.,  
H. LUGHTON KESTIVEN, D.Sc., M.D., Ch.M.  
Brighton,  
Queensland,  
October 8, 1957.

#### PENALTY OF ISOLATIONISM.

SIR: In an editorial entitled "Penalty of Isolationism" in your issue for July 13, page 60, I note the sentence "... perhaps the greatest hope lies in the adoption of a *lingua franca* for exposition and debate".

Perhaps you should call the attention of your readers to the fact that such a *lingua franca* exists and is already in use in more than ten American medical periodicals. Examples illustrating the use of this new "Latin without inflections" can be found in every issue of *Blood, Circulation, Circulation Research, The American Heart Journal, The American Journal of the Medical Sciences*, and others. It is easily read by educated people with either a Germanic or a Latin background, and lends itself easily to the expression of the most technical facts of science from mathematics to medicine.

<sup>1</sup> Minutes of Evidence to the Royal Commission (published in daily parts), First Day to Thirty-First Day, 1954, H. M. Stationery Office, London.

Some reasons why this wonderful development should be encouraged are given in the Sixth John Stanley Coulter Memorial Lecture, "Some Problems of Communication in Medicine", in the *Archives of Physical Medicine and Rehabilitation*, 38: 11-17, January, 1957. An example is also given there. Details of its history, grammar and vocabulary can be obtained from Dr. Alexander Gode, Science Service, Interlingua Division, 1719 N. Street NW, Washington 6, District of Columbia, U.S.A.

Interlingua is not the product of a single mind, but the result of concerted effort; it is easy to read because it is psychologically natural, and people who admired the spirit but deplored the difficulty of Esperanto find themselves enthusiastic about Interlingua.

Yours, etc.,  
FREDERIC T. JUNG, M.D.  
521 Ridge Avenue,  
Evanston, Illinois,  
U.S.A.  
September 5, 1957.

#### CONSIDERATIONS IN THE DIAGNOSIS OF A BREAST TUMOUR.

SIR: In these days of graphs and statistics, it is refreshing to read a purely clinical article, such as Dr. C. A. C. Leggett's (Journal, October 5), bearing the hall-marks of an acute and experienced observer. One is surprised, however, to read in the paragraph on discharge from the nipple the following: "A sero-sanguineous discharge associated with a definite tumour in the breast should be regarded as a breast neoplasm after the age of 30 years, and radical treatment is justified."

If this advice be followed, I believe that some of the breasts so widely removed would be found to contain nothing more sinister than cystic hyperplasia or duct papilloma, both of which are better treated by far less drastic surgery.

Nipple discharge is disappointing and misleading in diagnosis (except in the rare case where the microscope reveals pus or cancer cells). Its main value is due to the alarm it inspires in the patient's breast. It seems logical to treat such patients on the merits of the associated tumour, using adequate biopsy where doubt is felt.

In my view it is the woman with no demonstrable tumour who furnishes the problem, and I had hoped to learn how Dr. Leggett manages this group. Sir Cecil Wakeley<sup>1</sup> has written rather strongly on this subject.

Yours, etc.,  
J. M. YEATES.  
143 Macquarie Street,  
Sydney,  
October 16, 1957.

#### ANÆSTHETIC AND ALLIED CONSIDERATIONS IN RELATION TO CHILDREN'S ORTHOPÆDIC OPERATIONS.

SIR: In his article on the management of anaesthesia for orthopedic operations in children, W. H. J. Cole states that it is his practice to inject a solution of one in 250,000 adrenaline at the operation site while trichlorethylene is being inhaled (M. J. AUSTRALIA, October 5, 1957). This combination carries a considerable risk of inducing a serious arrhythmia, which may lead on to cardiac arrest.<sup>2</sup> I feel that the attention of anaesthetists should be drawn to the potential dangers of this technique.

Yours, etc.,  
MARGARET PATTERSON, M.B., B.S., D.A.  
58 Khandallah Road,  
Wellington, N.Z.,  
New Zealand.  
October 17, 1957.

#### PERIODIC HEADACHE ASSOCIATED WITH CEREBRAL ANEURYSM: REPORT OF A CASE SUCCESSFULLY TREATED BY SURGERY.

SIR: Dr. Geoffrey Vanderfield's account of lasting relief from migraine by the successful treatment of cerebral aneurysm makes interesting but provocative reading. I believe that true migraine is not a symptom of cerebral

<sup>1</sup> *Lancet*, 1947, 1: 62 (January 11).

<sup>2</sup> Langton Hewer, "Recent Advances in Anaesthesia", 1957.

aneurysm, and I think it is important that Dr. Vanderfield's patient presented, not with migraine, but with the symptoms of threatened rupture of an aneurysm. For that reason angiography and operation were so successfully performed.

An acute episode of ophthalmoplegia associated with severe headache, however, and "sick headache" for 17 years scarcely suggest the same etiology. Was the association of the two illnesses conclusively one of cause and effect as the writer states? That is interesting to postulate, especially in view of the relief from periodic headache that has followed craniotomy. But it should be borne in mind that the scalp flap described in your article divides the superficial temporal arteries, which is one of the procedures used to treat migraine.

It can be tempting to frequently recommend angiography for migraine, but it is of very doubtful value unless localizing symptoms or focal signs are present also. As Dr. Vanderfield says in his conclusion, angiography is indicated when other grounds for suspecting cerebral aneurysm or angioma also exist. I would add that in the absence of the established indications for surgery, an operation that either clips or excises such an anomaly may result in no improvement of the migraine symptom.

Yours, etc.,

STEVENS DIMANT, M.B., B.S., F.R.C.S.

Neurology and Neurologic Surgery,  
Puget Sound Medical Building,  
Tacoma 5,  
Washington,  
U.S.A.

October 14, 1957.

## Research.

### THE LIFE INSURANCE MEDICAL RESEARCH FUND OF AUSTRALIA AND NEW ZEALAND.

THE amount of the 1957 awards under the Life Insurance Medical Research Fund of Australia and New Zealand has been increased by £10,000 to a total of £35,000. The increased amount is a measure of the growing extent and importance of research into diseases of the heart and circulation in both Australia and New Zealand. The fund was established

five years ago to support research in this field, and this year's awards bring the total allocations in this period to £135,000. The awards for 1957 comprise ten grants-in-aid to research departments and three research fellowships, as follows:

#### Grants-in-Aid.

Baker Medical Research Institute, Alfred Hospital, Melbourne—for further research, under the direction of Dr. T. E. Lowe, into the biochemical aspects of energy production in the myocardium.

Department of Pathology, University of Melbourne—for further research, under the direction of Professor E. S. J. King, into dissecting aneurysm of the aorta.

Department of Physiology, University of Sydney—for further research, under the direction of Professor P. O. Bishop and Dr. P. I. Korner, into the haemodynamics of valvular incompetence.

Department of Pathology, University of Sydney—for further research, under the direction of Professor F. R. Magarey, into the pathogenesis of atherosclerosis.

Department of Pharmacology, University of Sydney—for further research, under the direction of Professor R. H. Thorp and Dr. E. A. Johnson, into the effect of drugs on the membrane resting and action potentials of single cardiac muscle fibres.

Cardiac Department, Royal Melbourne Hospital—for further research, under the direction of Dr. A. J. Goble, into the differentiation between aortic myocardial infarction and ischaemia.

Diagnostic and Metabolic Unit, Alfred Hospital, Melbourne—for research, under the direction of Dr. J. Bornstein, into the metabolism of the normal and diabetic arterial wall.

Department of Medicine, University of Melbourne—for research, under the direction of Professor R. R. H. Lovell, into the relationship between blood pressure and blood flow.

Department of Surgery, University of Sydney—for an investigation, under the direction of Professor John Loewenthal, into peripheral ischaemic states and their management.

Unit of Clinical Investigation, Royal North Shore Hospital, Sydney—for research, under the direction of Dr. Ian Monk, into the production and correction of mitral and tricuspid incompetence.

#### DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED OCTOBER 19, 1957.<sup>1</sup>

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism .. ..	3(1)	3(2)	8(3)	..	2	..	1	..	17
Amoebiasis .. .. .	..	..	..	..	1(1)	..	..	..	1
Ancylostomiasis .. ..	2	..	4	..	..	..	..	..	6
Anthrax .. .. .	..	..	..	..	..	..	..	..	..
Bilharziasis .. .. .	..	..	..	..	..	..	..	..	..
Brucellosis .. .. .	..	1	..	..	..	..	..	..	1
Cholera .. .. .	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus) .. ..	..	..	..	..	..	..	..	..	..
Dengue .. .. .	..	..	..	..	..	..	..	..	..
Diarrhoea (Infantile) ..	3(1)	5(4)	1(1)	..	3	..	3	..	15
Diphtheria .. .. .	..	2(2)	..	..	..	..	..	..	2
Dysentery (Bacillary) ..	..	..	1	..	..	..	..	..	1
Encephalitis .. .. .	..	1(1)	..	..	..	..	..	..	1
Filariasis .. .. .	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid .. .. .	..	..	..	..	..	..	..	..	..
Infective Hepatitis .. ..	51(29)	18(11)	2(1)	1	16(2)	..	..	1	89
Lead Poisoning .. .. .	..	..	..	..	..	..	..	..	..
Leptos .. .. .	..	..	..	..	..	..	..	..	..
Leptospirosis .. .. .	..	..	..	..	1	..	..	..	1
Malaria .. .. .	..	..	1	..	..	..	2	..	3
Meningococcal Infection ..	1(1)	..	1	..	1(1)	..	..	..	3
Ophthalmia .. .. .	..	..	..	..	..	..	..	..	..
Ornithosis .. .. .	..	..	..	..	..	..	..	..	..
Paratyphoid .. .. .	..	1	..	..	..	..	..	..	1
Plague .. .. .	..	..	..	..	..	..	..	..	..
Polomyelitis .. .. .	..	..	..	..	..	..	..	..	..
Puerperal Fever .. .. .	..	..	3	..	..	..	..	..	3
Rubella .. .. .	..	49(35)	8(6)	21(10)	14(14)	..	..	..	92
Salmonella Infection .. ..	..	..	..	..	..	..	..	..	..
Scarlet Fever .. .. .	4(3)	12(11)	4(2)	2(2)	2(2)	1	..	1	26
Smallpox .. .. .	..	..	..	..	..	..	..	..	..
Tetanus .. .. .	..	..	..	..	1	..	4	1	6
Trachoma .. .. .	..	..	..	..	..	..	..	..	..
Trichinosis .. .. .	..	..	..	..	..	..	..	..	..
Tuberculosis .. .. .	50(13)	19(12)	36(21)	7(7)	8(6)	3	..	..	123
Typhoid Fever .. .. .	..	..	..	..	..	..	..	..	..
Typhus (Flea-, Mite- and Tick-borne) .. .. .	..	..	1	..	..	..	..	..	1
Typhus (Louse-borne) .. ..	..	..	..	..	..	..	..	..	..
Yellow Fever .. .. .	..	..	..	..	..	..	..	..	..

<sup>1</sup> Figures in parentheses are those for the metropolitan area.

### Research Fellowships.

Dr. K. L. Cotton: A Travelling Fellowship for three years for research into chronic *cor pulmonale* and pulmonary hypertension at the Postgraduate Medical School, Hammer-smith, London.

Dr. M. J. Rand: A Travelling Fellowship for two years for studies on the physiology and pharmacology of cardiac function at the Department of Pharmacology, Oxford.

Dr. J. Margolis: A Research Fellowship for one year for investigation of the injury reaction initiated by the contact of blood with foreign surfaces at the Institute of Pathology, Royal Alexandra Hospital for Children, Sydney.

### EXPERIMENTAL RESEARCH INTO PROBLEMS OF AGING.

CANDIDATES wishing to submit entries for the 1958 Ciba Foundation Awards for papers descriptive of research relevant to basic problems of aging are reminded that these must reach the Ciba Foundation not later than January 1, 1958. Information about the awards, for those not already aware of the conditions, may be obtained on application from G. E. W. Wolstenholme, Director, and Secretary to the Executive Council, Ciba Foundation, 41 Portland Place, London, W.1.

## The Royal Australasian College of Physicians.

### ADMISSION OF MEMBERS.

At a meeting of the Council of The Royal Australasian College of Physicians held in Sydney on October 15, 1957, the following candidates, who were successful at an examination by the Australian Board of Censors, were admitted to membership of the College: Dr. W. G. Grigor, Dr. D. A. Handley, Dr. B. R. M. Hurt, Dr. J. McRae and Dr. R. A. Mellick, of New South Wales; Dr. G. W. Crook, Dr. D. J. Fone, Dr. N. B. Pinkus, Dr. B. A. Smithurst and Dr. G. R. Wagner, of Victoria; Dr. B. T. Emmerson, Dr. G. A. Hocker and Dr. K. J. Murphy, of Queensland; Dr. P. R. Hodge and Dr. S. Posen, of South Australia; Dr. H. J. H. Colebatch and Dr. A. M. Rankin, of Western Australia; Dr. J. C. H. Morris, of Tasmania. Dr. R. W. Hawker, of Queensland, was admitted under the special provisions of Article 37.

## College of General Practitioners.

### VICTORIA FACULTY.

#### Clinical Meeting at Prince Henry's Hospital.

The honorary staff of Prince Henry's Hospital, Melbourne, in conjunction with The College of General Practitioners, Victoria Faculty, are holding a clinical evening especially devoted to items of a practical nature for general practitioners on Thursday, November 28, 1957, at 8.15 p.m. All members of the British Medical Association are invited to attend.

### Nominations and Elections.

THE undermentioned have been elected as members of the New South Wales Branch of the British Medical Association: Douglas, Gerald Leslie, M.B., B.S., 1957 (Univ. Sydney); Chaffer, Ethel Valerie, M.B., B.S., 1947 (Univ. Sydney); Dawes, Peter Douglas, M.B., B.S., 1954 (Univ. Sydney); Murray, John Leslie Moreton, M.B., B.S., 1946 (Univ. Sydney); Swan, David Chetham, M.B., B.S., 1954 (Univ. Sydney); Barat, Tibor Ernest, M.D., 1933 (Univ. Budapest); Gonszka, Jacob, M.D., 1921 (Univ. Cracow); Szasz, Zoltan, M.D., 1915 (Univ. Budapest).

The undermentioned have been elected as members of the South Australian Branch of the British Medical Association: Derrington, Arnold W., M.B., B.S., 1947 (Univ. Adelaide); Miller, John Milton, M.B., B.S., 1956 (Univ. Adelaide); Wibberley, David John, M.B., B.S., 1955 (Univ. Adelaide).

## Medical Appointments.

Dr. P. J. Parsons has been appointed a member of the Anti-Cancer Council of Victoria as a nominee of The Royal Australasian College of Physicians.

## Deaths.

THE following deaths have been announced:

POOLE.—Gabriel Rotchfort Ruscombe Poole, on September 27, 1957, at Nambour, Queensland.

McDOWALL.—Valentine McDowall, on October 22, 1957, at Brisbane.

DALBY-HALL.—Maida Elsie Wilhelmina Buxton Daley-Hall, on October 28, 1957, at Rose Bay, New South Wales.

## Diary for the Month.

Nov. 12.—New South Wales Branch, B.M.A.: Executive and Finance Committee.  
Nov. 13.—Victorian Branch, B.M.A.: Branch Meeting.  
Nov. 18.—Victorian Branch, B.M.A.: Finance Subcommittee.

## Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

**New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney):** All contract practice appointments in New South Wales. Anti-Tuberculosis Association of New South Wales.

**Queensland Branch (Honorary Secretary, 33 L'Estrange Terrace, Kelvin Grove, Brisbane, W.1):** All applicants for Queensland State Government Insurance Office positions are advised to communicate with the Honorary Secretary of the Branch before accepting posts.

**South Australian Branch (Honorary Secretary, 80 Brougham Place, North Adelaide):** All contract practice appointments in South Australia.

## Editorial Notices.

ALL articles submitted for publication in this Journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given: surname of author, initials of author, year, full title of article, name of journal, volume, number of first page of the article. The abbreviations used for the titles of journals are those adopted by the Quarterly Cumulative Index Medicus. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary is stated.

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